


COLUMBIA LIBRARIES OFFSITE
HEALTH SCIENCES STANDARD



HX00047830

RECAP



Digitized by the Internet Archive
in 2010 with funding from
Open Knowledge Commons

CONTRIBUTORS TO VOLUME V.

BROWN, THOMAS R., M.D.

FRIEDENWALD, JULIUS, M.D.

KELLY, A. O. J., M.D.

McCRAE, JOHN, M.B. (Tor.), M.R.C.P. (Lond.).

MARTIN, CHARLES F., M.D.

OPIE, EUGENE L., M.D.

RIESMAN, DAVID, M.D.

ROLLESTON, HUMPHRY DAVY, M.A., M.D. (Cantab.), F.R.C.P.

STENGEL, ALFRED, M.D.

STOCKTON, CHARLES G., M.D.

MODERN MEDICINE

ITS THEORY AND PRACTICE

IN ORIGINAL CONTRIBUTIONS BY AMERICAN AND
FOREIGN AUTHORS

EDITED BY

WILLIAM OSLER, M.D.

REGIUS PROFESSOR OF MEDICINE IN OXFORD UNIVERSITY, ENGLAND; HONORARY PROFESSOR OF MEDICINE IN
THE JOHNS HOPKINS UNIVERSITY, BALTIMORE; FORMERLY PROFESSOR OF CLINICAL MEDICINE IN
THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA, AND OF THE INSTITUTES OF MEDICINE
IN MCGILL UNIVERSITY, MONTREAL, CANADA

ASSISTED BY

THOMAS MCCRAE, M.D.

ASSOCIATE PROFESSOR OF MEDICINE AND CLINICAL THERAPEUTICS IN THE JOHNS HOPKINS UNIVERSITY,
BALTIMORE; FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON

VOLUME V

DISEASES OF THE ALIMENTARY TRACT

ILLUSTRATED



PHILADELPHIA AND NEW YORK

LEA & FEBIGER

1908

Entered according to the Act of Congress, in the year 1908, by
LEA & FEBIGER
in the Office of the Librarian of Congress. All rights reserved.

7241

2-3

1907

U.S.

CONTRIBUTORS TO VOLUME V.

- THOMAS R. BROWN, M.D.,
Associate in Medicine in the Johns Hopkins Medical School, Baltimore, Md.
- JULIUS FRIEDENWALD, M.D.,
Professor of Diseases of the Stomach in the College of Physicians and Surgeons,
Baltimore, Md.
- A. O. J. KELLY, M.D.,
Assistant Professor of Medicine in the University of Pennsylvania; Professor
of the Theory and Practice of Medicine in the University of Vermont;
Professor of Pathology in the Woman's Medical College of Pennsylvania,
Philadelphia.
- JOHN McCRAE, M.B. (TOR.), M.R.C.P. (LOND.),
Lecturer in Pathology, McGill University, Montreal, Canada.
- C. F. MARTIN, M.D.,
Assistant Professor of Medicine and Clinical Medicine, McGill University,
Montreal, Canada.
- EUGENE L. OPIE, M.D.,
Member of the Rockefeller Institute for Medical Research, New York City;
Visiting Physician to the Presbyterian Hospital, New York City.
- DAVID RIESMAN, M.D.,
Associate in Medicine in the Medical Department of the University of Pennsylv-
ania; Professor of Clinical Medicine in the Philadelphia Polyclinic and
College for Graduates in Medicine, Philadelphia.
- HUMPHRY DAVY ROLLESTON, M.A., M.D. (CANTAB.), F.R.C.P.,
Senior Physician to St. George's Hospital; Physician to the Victoria Hospital
for Children, London; and sometime fellow of St. John's College, Cambridge,
England.
- ALFRED STENGEL, M.D.,
Professor of Clinical Medicine in the Medical Department of the University of
Pennsylvania, Philadelphia.
- CHARLES G. STOCKTON, M.D.,
Professor of the Principles and Practice of Medicine and Clinical Medicine in
the Medical Department of the University of Buffalo, Buffalo, N. Y.

CONTENTS OF VOLUME V.

DISEASES OF THE ALIMENTARY TRACT.

CHAPTER I.

INTRODUCTORY DISCUSSION ON THE DISEASES OF THE DIGESTIVE APPARATUS	17
By CHARLES G. STOCKTON, M.D.	

CHAPTER II.

DISEASES OF THE MOUTH AND SALIVARY GLANDS	39
By DAVID RIESMAN, M.D.	

CHAPTER III.

DISEASES OF THE ŒSOPHAGUS	77
By JOHN McCRAE, M.B. (Tor.), M.R.C.P. (Lond.)	

CHAPTER IV.

FUNCTIONAL DISEASES OF THE STOMACH	105
By JULIUS FRIEDENWALD, M.D.	

CHAPTER V.

ORGANIC DISEASES OF THE STOMACH	175
By CHARLES F. MARTIN, M.D.	

CHAPTER VI.

DISEASES OF THE INTESTINES	327
By ALFRED STENGEL, M.D.	

CHAPTER VII.

DISEASES OF THE PERITONEUM	495
By HUMPHRY DAVY ROLLESTON, M.A., M.D. (CANTAB.), F.R.C.P.	

CHAPTER VIII.

- SPLANCHNOPTOSIS—ENTEROPTOSIS—GLÉNARD'S DISEASE . . . 599
By THOMAS R. BROWN, M.D.

CHAPTER IX.

- DISEASES OF THE PANCREAS 621
By EUGENE L. OPIE, M.D.

CHAPTER X.

- DISEASES OF THE LIVER, GALL-BLADDER, AND BILIARY DUCTS 683
By A. O. J. KELLY, M.D.

DISEASES OF THE ALIMENTARY TRACT.

CHAPTER I.

INTRODUCTORY DISCUSSION ON THE DISEASES OF THE DIGESTIVE APPARATUS.

By CHARLES G. STOCKTON, M.D.

IN this introduction it is intended to discuss certain critical relations that may exist between disturbed physiological activity and structural changes as expressed in morbid processes. The subject is divided under the following headings:

1. The Whole and the Part.
2. Individuality in Disease.
3. The Interrelation of Digestive Functions.
4. Adaptability and Vicarious Action of the Digestive Functions.
5. The Relatively Great Importance of Motor Functions.
6. The Nature of Functional Disturbances of Digestion.
7. Functional Disturbances in Relation to Structural Disease.
8. Structural Changes Resulting from Functional Disorders.
9. Structure and Function in Relation to Peptic Ulcer.
10. The Question of Secretion.
11. Infection in Relation to Digestion.
12. Visceral Blood Supply in Relation to Digestion.
13. False Combinations of Secretion—Disharmony.
14. The Place of the Biliary Tract in Digestive Pathology.
15. Some Peculiarities of Pancreatitis.
16. Possible Correlation of the Abdominal Viscera through the Sympathetic.
17. Splanchnoptosis and Overtension of the Peritoneum.
18. Psychoneuroses in Relation to Digestion.
19. Variations in the Intestinal Flora in their Relation to Diseases of the Digestive Tract.

1. **The Whole and the Part.**—We are accustomed to look upon digestion as the function of an extensive and complicated apparatus, but it is so intimately related with the functions of other parts of the body that it is difficult to draw lines of precise limitation beyond which it can be said the digestion has no part and from which it receives no reciprocal activity. We commonly speak of the digestive apparatus as including the alimentary tract and those important glands which contribute specific secretions to the

advancement of digestion; but as absorption and assimilation, on the one hand, and the formation and withdrawal of catabolic and waste products, on the other, are so immediately related to the preliminary digestion, it is impossible to form a clear conception of the diseases of the digestive organs without taking into consideration the state of other and contributing parts of the organism. While it makes for simplicity in description to exclude from implication those organs not commonly grouped with the digestive apparatus, this does not result in a correct understanding, and, therefore, if one is to find an explanation not only for a disturbed physiological state, but also, in instances, for structural changes in the digestive organs, he must widen the field and direct his study to the state of the nervous system, including its psychical manifestations, to the fluids of the body, to the blood pressure, the metabolism of the tissue, and to the state of the excretories. The importance of these matters is perhaps unconsciously recognized when we declare that good digestion depends upon restful sleep, fresh air, sunlight, physical exercise, and the proper activity of the bowels, kidneys, and skin. When we disregard these essential matters, it is difficult to form a just conception of the nature of digestive disturbances, or to prescribe successfully therapeutic measures for their relief. For this reason one may truly say of a given dyspeptic, the trouble arises in the brain, or in the lungs, the heart, the kidneys, etc. The pathology of stomach diseases is not limited to that organ, but is the expression there of disturbances that may be widely distributed through the whole body. This fact applies to other parts as well as to the stomach. Answering Virchow's question, "Where is the disease?" we have sought to give each disease a local habitation, but this is only a relatively justifiable conception as regards any disease, although more true of some than others. There are probably no diseases referable solely to the kidneys, the heart, or the blood; the man is sick, and it appears here or there.

Although a large proportion of the affections of the digestive apparatus are functional, many cases with which we are confronted present an association of functional and structural abnormalities. Congenital malformations, displacements of abdominal viscera, and hernia, both internal and external, are structural defects. The infiltrations like amyloid disease, the more specific infections like cholera, typhoid fever, amœbic dysentery, and tuberculosis must be regarded as beginning their effects upon the body through their action upon its structure. But almost immediately they lead to functional disturbance, both local and general, and it will therefore be understood that the state of general vitality of the patient conditions the functional activity of each organ, and is a potent factor in the outcome of any local disease.

Many facts relating to digestion have come into our possession during the past twenty years, but the mere accumulation of facts and their presentation in isolation is not the most satisfactory method of studying a subject. By what laws are these facts related? In what is the favorable activity of one organ dependent upon that of another? Of the various links in the digestive chain, which are the more indispensable? If we shunt the action of certain parts through surgical intervention, what will be the final result in the organism? These are questions of vital importance, and by arranging our data with a view to answering such inquiries we throw most light upon the subject as a whole.

2. Individuality in Disease.—It is necessary to value carefully the individual equation. On the one hand misconception may arise when generalizations are based on the manifestations of a single case. On the other hand the question of individual peculiarity and idiosyncrasy must be carefully weighed, so that we may avoid the feudality of classification and the tyranny of established rule. For instance, von Noorden has shown that certain starches are comparatively successful foods in particular diabetics, although quite the reverse is true in other patients with this disease. Through idiosyncrasy the eating of potatoes or the flour of certain grains, although usually acceptable, invariably excites intense gastro-intestinal distress in an occasional individual. This principle applies to other matters than food. The sensitiveness of some persons to the ill effects of mental excitement, physical fatigue, or exposure to cold must not be overlooked, nor must it be cast into a rule. Doubtless there are many individual factors that are at times active in producing physiological derangements which are as yet undetected.

3. The Interrelation of Digestive Functions.—Common experience has taught us that the mental state and the sense of taste in some unknown way affect the primary digestion. Thus worry induces leanness, and happiness favors the opposite condition, in a measure independently of the amount of food taken. Some of the steps by means of which these effects are obtained are explained by the work of Pawlow. The digestive secretions and the motor activity of the stomach and intestine are excited by proper psychical stimulation, and some of them are inhibited, others overexcited, by distress. Hirsch and von Mering have established the fact of an intimate relationship between gastric secretion and motion, and the secretions of the duodenum, pancreas, and liver. The presence of acid gastric juice in the duodenum leads, through reflexes arising in the latter, to the closure of the pylorus, preventing temporarily a further escape of gastric contents. This gastric juice within the duodenum, until neutralized by other secretions, stimulates the secretion of pancreatic juice, bile, and succus entericus; thereupon the pylorus again opens and the rhythmic movements of the stomach expel a further portion of the gastric juice into the duodenum, which, in turn, leads again to the closure of the pylorus. This reciprocal action demonstrates the important relation of one part of the digestive apparatus with another. Bayliss and Starling have shown that the presence of acid gastric juice in the duodenum stimulates the secretion of secretin, a substance which, passing into the blood and reaching the pancreas, excites in that organ its specific secretion; so it would seem that the intermittent and regular discharge of acid gastric juice into the duodenum is indirectly the normal stimulus of the pancreatic secretion. Trypsin, which was formerly supposed to be a secretion of the pancreas, has been shown by Pawlow and his students to be the result of the action of a substance found in the succus entericus, known as enterokinase, acting upon trypsinogen, which is secreted by the pancreas. It is by a combination of these two that trypsin is formed.

Attention has been called to the fact that the psychical condition may influence salivation and gastric secretion; then there follows a stimulation of the motor activity of the stomach and intestine, of the secretions of the duodenum, the pancreas, and the liver, and finally, the formation of at least one substance that activates the secretion of the pancreas and other organs which otherwise would remain relatively passive.

But the impulse starting in a psychic state does not end in its effects upon the liver and pancreas. The functional activity of the small intestine is greatly influenced by the presence therein of the right proportion of secretion from the liver, pancreas, and other glands, and intestinal digestion would suffer in case of decrease or derangement in this supply. Under such conditions irritation of the intestinal mucosa, disturbance of its motor function, and lowering of the resistance of the part would result. With such a state of affairs it would be expected that the bacterial life, especially within the gut, would be multiplied, fermentation occur, and infection of the organism would be facilitated.

With physiological economy, there goes on in health the absorption of biliary salts and probably other substances by the intestine in order to maintain a proper balance of the organism. When there exists a marked disturbance of intestinal digestion we have reason to suppose that it interferes with this absorption. While the liver may be embarrassed through not receiving by way of the portal circulation the usual supply of recovered substances, it suffers at the same time because of the absorption of the products of putrefaction. It has also been shown by Adami that living bacteria reach the liver from the intestine.

When the liver is thus overtaxed, a general toxemia supervenes, the whole body is injured, and hæmolysis is often exaggerated. There exists a remarkable, although somewhat obscure, relationship between the liver and the kidneys, and under conditions that are herein described renal elimination may be decreased or otherwise deranged. To these evils others may be added when the colon, suffering in turn, contributes to the general toxæmia. Delafield has emphasized the frequency of chronic colitis as the result of interstitial nephritis, and others have recognized the injurious effects of colitis upon the kidneys.

Relatively great importance attaches to that portion of the digestive apparatus which includes the pylorus, duodenum, and pancreas. A fuller acquaintance with the functions of these organs not only throws light on the clinical course of the digestive diseases, but serves to warn us that surgical intervention must take into account the possible harm that may result from any great disturbance of the relations of these parts.

4. **Adaptability and Vicarious Action of the Digestive Functions.**

—At the same time we are impressed by the facility with which one portion of the alimentary tract is able to dispense with the assistance which it ordinarily receives from another, and by the success with which nature adapts herself to the absence of the function of the stomach and of large portions of either the small intestine or colon. With complete atrophy of the gastric mucosa, or with the bile diverted through a fistula from its intestinal course, or with the pancreas made practically functionless through chronic inflammation, the patient may survive and maintain a moderate degree of general nutrition. The loss of the function of a single digestive organ may not be accompanied by as serious results as ensue from lowering of vitality in the apparatus generally, such as follow grave diseases of the nervous system, or the chronic intoxications and infections. In other words, the organism seems capable of sustaining almost in entirety the loss of function of certain of the digestive organs. Nutrition sometimes fails as the result of depression, affecting all parts of the apparatus at one time. But to reiterate, it would be a misapprehension to suppose that health can be perfect after the loss

of function of any one of the important digestive organs, or after the removal of parts, as, for instance, the stomach, by surgical intervention.

5. The Relatively Great Importance of Motor Functions.—Our understanding of digestive diseases has been much advanced by a clear conception of the role of motion in health and in disease as compared with that of secretion. It appears to the writer that the importance of motor disturbances of the digestive tract is not yet fully recognized. We find that although Nature adapts herself more or less successfully to the absence or derangement of what would seem to be necessary secretion, she immediately resents, and dangerous symptoms supervene upon, marked disturbances of motility, whether there is overexcitation or overdepression. Especially, we find that evil results are brought about by such interruption of motion as gives rise to stagnation of gastro-intestinal contents or the interruption of the flow of secretion from the great digestive glands. In the latter case the mischief is not so much from the retention of secretion as from the mere fact of stagnation and from the infections and intoxications which are thereby entailed. In a sense it may be said that an exception exists in the instance of the large intestine, where, in event of chronic constipation, we observe a considerable stagnation without necessarily serious consequences. This may be explained from the fact that it is the function of the lower part of the colon to retain its contents, and therefore in constipation we have to deal merely with the exaggerated function, going on ultimately to disease. But even here we recognize the evil consequences that result in some instances of even moderate stagnation, for constipation in certain individuals is immediately succeeded by general digestive derangement as well as by important constitutional symptoms. This is especially true of those unaccustomed to constipation or those in whom there is inadequate activity of the lungs, liver, and kidneys. In relation to the kidneys and liver, this statement is likely to pass unchallenged by some who would doubt its applicability to the lungs, but we probably fail to recognize the full importance of elimination by way of the lungs. In sufferers from emphysema, chronic fibroid pneumonia, or continued pulmonary congestion, the patient is strikingly relieved by timely stimulation of the skin, bowels, and kidneys. Doubtless this arises in part through forwarding the circulation and assisting oxygenation, but the practitioner may convince himself that the element of elimination must also be counted. Individuals long accustomed to atony of the colon may acquire a tolerance, and experience little inconvenience from prolonged constipation. This is but another illustration of the success with which the organism adapts itself to untoward conditions. At the same time it shows how intimate is the relationship between the digestive functions and those of remote apparatuses like the genito-urinary and the respiratory.

The studies of Cannon indicate that our conception of the nature of gastro-intestinal motion has to be revised, and it is probable that new light will be thrown upon certain digestive derangements as a result.

6. The Nature of Functional Disturbances of Digestion.—The nature of the so-called functional diseases is better apprehended by studying them along more general lines such as those already suggested. The tangled symptomatology is easily misunderstood, and, for a long time, the pathology and the pathogenesis for the most part evaded the clinician. So far as relates to the stomach, a better conception of the facts followed soon after Leube's adaptation of Kussmaul's stomach tube as an instrument of diagnosis. It

was then seen that the state giving rise to a complex of symptoms formerly described as "indigestion" was not a single pathological entity, that it was in fact not one thing, but a varied derangement of gastric activity, the more or less direct result of irritation or depression of the sympathetic nervous system. Therefore, these functional affections were early divided into two classes: first, functional disease from excitation, and second, from depression. This division, while in a sense well justified, is not so simple as it appears; for, in point of fact, it is not always practicable to decide in which group a given disturbance shall be placed. Besides, in not a few patients we may discover evidences of an irritative character at one time and depressive at another. Nevertheless, the classification is useful and helpful to understanding. It was soon perceived that symptoms from excitement might not materially differ from those following depression, and that both are manifestations of physiological fatigue, whether arising from particular or general sources of exhaustion. These different, varying manifestations were soon divided into three classes and termed disorders of secretion, motion, and sensation. Now, while this is a natural and probably permanent step in classification, it must be materially modified and supplemented in order to make it of value in the understanding of the actual disease. True, we do occasionally meet with a disturbance that seems to be wholly motor, secretory, or sensory, as, for instance, cardiospasm or gastralgia; but, as a rule, and with relatively few exceptions, we find a complex in which two or all of these divisions of symptoms occur simultaneously. Illustration may be found in most of the cases of so-called gastric atony.

The trend of events that makes up a definite and more or less constant group is to be recognized, and the complex identified and named only after a careful study of the situation. This study must not be limited to the mere investigation of the functional behavior of the stomach. Commonly, indeed almost invariably, the etiology of the trouble will be found in some remote and perhaps unexpected region of the organism, to some leak of general energy, if the expression is permissible, to some undiscovered irritation of the nervous system. Thus a retroverted uterus, proctitis, or a displaced kidney may indirectly lead to important digestive disturbances, but more frequently causes of gastric asthenia are to be found in eyestrain. This subject has been so widely discussed in America, and from so many points of view, that it is somewhat threadbare; yet its signal importance remains largely disregarded. Irregular or asymmetrical astigmatism is the visual defect most often responsible for the functional disturbance, but it is not always in astigmatism of high degree that the trouble arises. It is more commonly found in instances of moderate degree of astigmatism with axes differing in the two eyes, and especially in anisometropia. Although not limited to that period of life, the nervous disturbances following these visual defects are apt to appear after the age of maturity, and are especially active when the vitreous body begins from age to lose in pliability. We are indebted to Gould for insisting upon the reality of the matter.

Formerly our text-books referred to but few gastric disorders save the well-established structural processes. With the advent of newer methods of study, as taught us by Riegel, Ewald, Dujardin Beaumetz, and others, there arose a tendency to multiply the number of gastric neuroses until the array of titles in a modern book on stomach diseases becomes somewhat bewildering. Undoubtedly much good has come from this minute investigation of

functional disorders of the stomach. Among other advantages, it has added to our knowledge of the physiology, but now that so many years of conscientious work have been paid out and so many valuable facts have been gathered in, we are in an admirable position to review the field and to arrange the material along the lines that experience and a sense of proportion would direct.

When this has been well done we shall doubtless agree that our conception of stomach troubles has undergone an absolute reconstruction since the days when text-books treated of "indigestion" and "flatulent dyspepsia;" but we shall admit also that there has been something of overrefinement in the classification, description, and management of the gastric neuroses. It is rather remarkable that this outgrowth of recognized gastric neuroses should have occurred during an epoch in which a basis in definite morbid anatomy was required of practically all diseases; and when no structural abnormality was shown to bear a constant relation to a symptom-complex, the pathogenesis of that disease was held in doubt. Notwithstanding this, our assembly of nervous affections of the stomach, for which no morbid structural setting was found, was adopted into our nomenclature and was not disputed by most of our clinicians.

Hereafter, as now, we shall doubtless admit that this was a decided advance; but already we are discovering that we had in several instances misconceived the real meaning of the situation. In accepting the doctrine that we might have an array of motor, secondary, or sensory disturbances merely as the expression of a neurosis, we became too oblivious of the fact that this supposed neurasthenia might in fact rest upon some definite local disease that escaped detection.

7. Functional Disturbances in Relation to Structural Disease.—

This oscillation of opinion that makes for a resting in the truth has come in part as a result of the surgical treatment of abdominal diseases. It was seen that after the removal of gallstones and the drainage or excision of the gall-bladder, not a few of the supposed neuroses promptly disappeared, and the physician who had rested satisfied with the diagnosis of hyperchlorhydria was disconcerted. Probably we have yet to learn the full number and the importance of the gastric phenomena that may develop as the result of irritation in the gall-bladder and its vicinity. We are already aware that this irritation occasions gastric hyperæsthesia, hyperchlorhydria, and hypersecretion, besides the syndrome of transient pyloric obstruction with characteristic delay in emptying the stomach and that well-known train of symptoms which comes from food stagnation. This latter event may be encountered in cases in which there is no actual stenosis, no structural narrowing of the pylorus or duodenum, but, as pointed out by Soudpult and others, merely a reflex or spasmodic affection of the pylorus, with or without coincident derangement of gastric secretion, but, necessarily, with serious digestive disturbance both in the stomach and duodenum.

We have long known that this train of events was a natural accompaniment of pyloric or duodenal ulcer. Now that cholecystitis is admitted as a cause, there is difficulty in persuading some men to believe that pyloric spasm ever occurs as the result of a neurosis; on the contrary, they hold that there is always local irritation in the region of the pylorus if it could be found. They affirm the local cause and leave the burden of proof with those who yet hold the position that there remains a proportion of cases in which

the functional element is the only and sufficient cause. As the evidence that may be adduced in supporting this opinion has a fitting application to other supposedly functional disorders, it may be profitably mentioned at this point.

To begin with, only a neurotic origin has been able to explain a few of the reported cases of congenital pyloric stenosis. Then there is the analogy of spasm in the sphincter portion of other organs. The idiopathic dilatation of the cesophagus following cardiospasm which Meltzer has elucidated, is an example, to which may be added vesical and anal spasm, besides the involuntary contraction in laryngismus, vaginismus, etc. Still further evidence is found in the cure of these morbid contractions by measures directed solely toward calming excitability. Finally, the surgeons have learned from unfavorable experience the futility of attacking neurotic affections by operative measures, for the symptoms not only continue, but often increase thereafter. That there may remain some obscure and hidden structural defect is of course possible, but, in the light of all facts, improbable. Indeed, when we come to analyze the cases of pylorospasm secondary to a diseased gall-bladder, it will be found that some of them present no signs of former perigastritis nor extension of inflammation to the pylorus or duodenum. In these very cases, then, we must admit some indirect functional excitation, differing, it is true, from our conception of a neurosis, but leaving still something that morbid anatomy fails to explain.

An unprejudiced view of all sides of the question would seem to warrant our granting that a disordered nervous system may at times give rise to cardiospasm or pylorospasm, but the warning should be kept in mind that, except in comparatively manifest cases, we should seek the cause in some marked irritation at or near the vicinity of the abnormal contraction.

8 Structural Changes Resulting from Functional Disorders.—Accepting the doctrine that many gastric disturbances arise from nervous causes with and without assignable irritation at some point remote from the stomach, the question occurs as to how far it is possible for structural diseases of the stomach to follow as a secondary result of severe or prolonged insult to the nervous system. The belief has long been held that gastric atony and even dilatation of the stomach may ensue from long-continued nervous depression. It is not difficult to explain gastrectasis as the result of pyloric or upper intestinal obstruction, but there is a large residuum of cases of this affection in which no obstruction is found, and their origin is usually attributed to prolonged nervous depression and overtaxation of the stomach with resulting myasthenia. In this connection it is interesting to note the rapid accumulation of reported cases of acute dilatation of the stomach following surgical procedures. These instances of acute gastrectasis succeeding upon surgical shock are not limited to abdominal operations or injuries, for there are well-authenticated cases which have developed after extensive injury to one of the extremities, the joints, or subsequent to grave pneumonia, or accompanying acute tuberculosis or carcinoma. In a proportion of these cases a real obstruction is found, usually in the duodenum, occasioned by compression of the gut between the root of the mesentery and the spinal column, the result of dragging of the intestines downward. This occurs in some conditions involving great relaxation of the abdominal wall. In a majority of cases no obstruction or other causes save nervous shock has been discovered.

It would seem that an important lesson is to be learned from these cases. The fact that dilatation can occur within a few hours, with manifestations so serious that death usually results unless proper intervention is resorted to, and that this can occur without any direct injury to the stomach or other abdominal viscera, seems a striking illustration of the relationship that exists between innervation and the well-being of the digestive apparatus. If shock can induce such acute and serious structural change in the stomach, we may expect analogous behavior in the intestine and other parts. Until autopsy becomes more general, we are not likely to know what percentage of unexpected fatal terminations after surgical operations may depend upon this accident.

If acute gastrectasis can arise from great depression of visceral innervation, it is conceivable that depression of lesser degree and long continuance may result in atony; and if to this we add the element of overtaxation of the stomach from indiscreet eating, we may easily explain those cases of gastric atony which do not result from obstruction.

One who has had wide experience in the examination of gastric contents is aware of the striking influence which the state of the nervous system has upon gastric secretion. It is not uncommon to find instances of hyperchlorhydria which disappear as soon as the patient follows a quiet, orderly life. On the other hand, in hypochlorhydria systematic rest, with consequent improvement in the general health, often leads to the return of normal secretion. In many instances it will be found that the neurasthenic state that gives rise to the gastric symptoms may not depend upon inherited weakness nor upon general nervous exhaustion, but, as before mentioned, rather upon the overfatigue of some particular part of the nervous system. Mere local treatment of the stomach unaccompanied by improvement in other directions is usually unsuccessful in inducing a cure.

One naturally inquires whether these long-continued disturbances of secretion may not lead to inflammatory and other lesions of the gastric mucosa. Gastritis has been given extensive consideration in most treatises on stomach diseases, and when one recalls the importance of the affection, this may not seem out of place. According to the experience of some, the frequency of gastritis is exaggerated and such observers believe that the affection is in fact much less common than is usually claimed. Nevertheless, gastritis is a troublesome affection, and its etiology should be carefully looked into. The disease is to be regarded as an infection of the stomach. One is impressed, however, with the truth long ago pointed out by Beaumont, that the recuperative and regenerative power of the gastric mucosa is remarkable, and we find that not only inflammation, but moderate trauma of the stomach, goes on to rapid repair, provided the general conditions are favorable. What is usually regarded as gastritis is in fact a functional irritability of the stomach lining, a hyperæsthesia gastrica, and not a true inflammation. In the extremes of age, however, or when the resisting power of the organism has been greatly reduced by long-continued fatigue, general infection, or great nervous depression, experience shows that gastritis is readily excited, and that, when once present, it is likely to continue until the general health is improved. It is interesting to recall that this also is true of the gastric neuroses, and just as hyperæsthesia and motor irregularity may be caused and continued by systemic depression, so also may this be true of gastritis. Apparently a good deal of confusion exists in the mind

of the average practitioner as to when a case is inflammatory and when it is merely one of functional irritability. It is important to make the differentiation, but it is occasionally difficult, as also is the recognition of etiological factors upon which either affection is based. While it is not hard to understand how a functional disturbance may arise as a result of nervous depression or excitement, the view that gastritis may depend upon a similar train of circumstances is a view that has not gained sufficient recognition.

One must not dwell too much upon systemic depression as a cause in the development of gastritis; of course, it is only a factor which predisposes to infection. It is commonly and justly believed that the toxæmias also invite gastritis. This probably results from a lowering of vitality in the gastric mucosa such as the intoxications may induce in other tissues. Local disturbances like acute constipation, hepatic congestion, and overloading the stomach are factors which generally aid in setting up inflammatory reaction. The point should be made, however, that while one person easily withstands indiscretion in diet, another person under the same circumstances immediately suffers from gastritis, and this is true because of the lowered resistance to infections which exists in certain individuals, a truth that we find paralleled in the course of infections in other organs. Thus, in individuals who are predisposed to appendicitis, colitis, and cholecystitis, it is possible to find the explanation in reasoning along the lines here indicated.

9. Structure and Function in Relation to Peptic Ulcer.—In the search for the etiology of peptic ulcer we have discovered a number of other things, but not precisely that, and the ground has been so much tramped over that the boundaries of the question are considerably obscured. It is highly probable that when we include in one group cases found postmortem, those produced experimentally, and those recognized clinically, we are not talking about one and the same thing in each instance. Gastric ulcer has been produced in animals experimentally by prolonged feeding on infected food. Inferentially, the same result would obtain in man; nevertheless, except when stagnation exists, the stomach with peptic ulcer is remarkably free of the usual evidences of infection. Virchow apparently solved the problem by the hypothesis of thrombus, and this has been shown to be true of rare cases, while it apparently has no place as a cause in the great majority. A like conclusion may justly attach to the theory that gastric hyperacidity, lowered alkalinity of the blood, local trauma, the accidental manifestations of other diseases, like tuberculosis, syphilis, scurvy, or diabetes, are the direct and single cause. That the gastric mucosa may suffer ulceration like other surfaces of the body and from identical causes will probably not be disputed, but it is scarcely justifiable to regard all these as instances of peptic ulcer. Undoubtedly some features are common to all these ulcerative gastric lesions and the apparently identical processes observed in the duodenum, and rarely in the cardiac extremity of the œsophagus. These common features appear to arise from the peculiar and similar experiences to which these parts are subjected by the digesting and eroding action of the gastric juice upon tissue relatively devitalized by any cause whatever.

If we limit our attention wholly to these characteristics of gastric ulcer, namely, gastric hyperacidity and general lowering of the vitality of tissue, we fail to account for the special cause that may explain the peculiar characteristics of the peptic ulcer.

We should arrange and consider the following facts: (1) Classical

acute peptic ulcer is prone to occur in chlorotic young women, and, although the exceptions are many, the rule must not be obscured. (2) Classical chronic or indurated peptic ulcer is prone to occur in men past middle age. (3) Peptic ulcer is prone to localize itself either at the lesser curve on the posterior surface near the pylorus, or, as shown by Mayo, in the duodenum. (4) Its tendency is to continue, to become chronic. (5) Some ulcers show immediately a design to perforate, others seem forever limited to the mucosa. In acute ulcer there is nearly always present an overactivity in the secretion of very acid gastric juice.

In reviewing these special features we recall unquestioned instances tending to nullify the rule; but too much weight should not be given to the exceptions in a subject holding so many possibilities of confusion; we should rather attempt to trace the usual lines more deeply, while at the same time noting and interrogating the unusual. After doing this it would seem clear that in the gastric ulcer of the dead house we are dealing with several entities—differing in cause, tendency, history, and termination—although there are certain manifestations in which they are alike.

It would seem that there is some as yet unknown factor at work in the cause of true peptic ulcer, some factor which lowers the vitality of tissue, especially in certain regions of the stomach and duodenum. Apparently this factor is relatively common in early adult life in women, and after middle life in men, and in the latter is more disposed to chronicity. What is this factor? Will it be found in the local deficiency of antibodies, as suggested by Weinland? In local ischæmia? In preceding lesions of neurotrophic nature, analogous to herpes or to the perforating ulcer of the integument? These questions remain unanswered, and the operative treatment of gastric and duodenal ulcer, while proving that the disease is more frequent than we were prepared to admit, and showing that, in men, duodenal peptic ulcer is seen about as often as gastric ulcer, has not materially aided in explaining the nature of the process. The hypothetical disappearance of antibodies may well account for the loss of tissue, but how shall we explain the local deficiency in antibodies? May this not rest in the neurotrophic realm?

Whatever may be the exciting cause, we are reasonably certain that the increased activity of the gastric secretion and its overacidity contribute much toward the rapid evolution of the ulcer and toward its chronicity. The pain and gastric irritability are to be explained in part by the pyloric and gastric spasm and by the delay in the onward passage of chyme. Thus, the rationale of the usual medical treatment by rest, diet, alkalies, local sedatives, and external applications is made evident. When failure follows this treatment, the result would seem to be attributable largely to the fact that it requires the utmost attention and pains to prevent interruptions in the course of treatment, and that a short interruption may prove sufficient to overcome what has been gained by days of patient effort. Whether food is denied or in proper form is given frequently and persistently, the idea of keeping the gastric acidity low and overcoming the element of spasm must never be forgotten; even after gastro-enterostomy done to procure drainage, the benefit resulting from a modified diet and a lowered gastric acidity is of considerable importance.

By a control of the functional activity of the organ we are able to modify the course of the structural disease. Of course, this principle is not limited to gastric ulcer, but it seems to illustrate the important and persistent inter-

relation that exists between organs and functions. Perhaps we should be more earnest in tracking structural diseases to a starting place in functional derangement, to the finding of lowered resistance in some areas of tissue as the result of biochemical deficiency, possibly neurotrophic in its inception. All this may strike some men as lacking the evidence of acknowledged experience, but such a decision would be premature. There are many facts to be marshalled in its support. The bizarre and rather unaccountable manifestations grouped by Osler under the head of angioneurotic oedema should be recalled as an illustration of visceral lesions apparently secondary to neurotrophic disturbance.

We observe herpes facialis, sometimes persisting for weeks together, appearing in some individuals whenever the health is depreciated by fatigue or other general causes. Such eruptions are prone to attack a certain area, reappearing in the same place and occasionally accompanied by severe pain.

We occasionally observe perforating ulcer of the arm or leg persisting indefinitely, resisting topical treatment and recurring after excision. We should recall Raynaud's disease and also the curious non-traumatic hæmatoma auris, particularly observed in the insane. Marked subcutaneous extravasations of blood appear in certain neurotics in various parts of the body, disappearing, then recurring after a period of health. In one patient the writer observed this at intervals during several years, sometimes alternating with, sometimes accompanied by, the classical symptoms of gastric ulcer, and especially by hæmatemesis of grave character. In erythema multiforme one may observe the wide involvement of the mucous membrane, and one severe case was observed in which this disease was accompanied by the symptoms of gastric ulcer. This collection of presumably neurotic affections associated with structural lesions shows an indubitable relation between the nervous system and the integrity of the tissue.

We have insufficient experimental evidence to prove that either a nerve lesion or a neurotrophic disturbance is a frequent cause of peptic ulcer, but the behavior of the affection strongly suggests the possibility. These statements are intended to emphasize a principle that seems to be too little recognized in pathological processes, that of the very close relationship existing in all parts of the organism between the structure and the functions which utilize that structure.

This is a recurrence of the thoughts already expressed, that we are prone to think of structural disease as something separate and apart from functional disease. It is true that we often find a structural change to explain the supposed functional disturbance, but it seems equally true that the functional disturbance is the forerunner of an etiological factor in, sometimes the cause of, the structural disease.

The hyperchlorhydria so commonly present in peptic ulcer is often intensified by food retention, secondary to pyloric spasm, and the same cause is responsible for some cases of that condition usually called gastrosuccorrhœa. It must be admitted, however, that there are cases without over-retention from spasm, cases in which an excess of gastric juice is secreted; and whether this is or is not commonly true in ulcer, the fact remains that hyperchlorhydria is generally found. It was long believed that this might be produced by local stimulation occasioned by the open ulcer. Pawlow's observations tended to discredit this view. He failed to excite gastric secretion in animals by irritation locally applied, although juice flowed freely

at the sight of food and upon its introduction into the stomach. A highly acid secretion is made to flow in dogs as a result of faradization of the mucosa. Recently, A. Schiff has been able to excite the flow of gastric juice by local stimulation when the factor of psychic effects and the presence of food in the mouth or the stomach were eliminated.

Clinically, we find that the acidity is increased by feeding and even by the suggestion of eating, as demonstrated by Pawlow. But we also know that hyperchlorhydria is present in gastric ulcer even when the patient is fasting and when the psychic influence is wanting. It is probable, therefore, that gastric secretion is excited by the irritation of an ulcer as well as by the suggestion or presence of food in the stomach.

One may conclude that there is usually an excessive secretion and at times retention of gastric juice in this disease, and there can be no doubt of the unfavorable effect of the hyperacidity in the course of the ulcer. Further, we know that chlorosis, so often associated with ulcer, is generally accompanied by hyperchlorhydria, and that an impressionable nervous system at least predisposes to the oversecretion.

10. The Question of Secretion.—The behavior of the stomach in the matter of secretion presents a problem not yet satisfactorily elucidated. By the action of certain proteids upon the mucosa of the pyloric end of the stomach there is excited in the glandular structure of the part a substance which passes into the general circulation, returns to the gastric mucosa, and excites therein an active secretion of gastric juice. Pawlow and Popielski have shown that this secretion occurs after the destruction of all nerves connecting the stomach with the ganglionic centres. Edkins has shown that it is not the result of a local reflex in the gastric walls, but is owing to a chemical influence, in mode of action similar to that of secretin and other so-called hormones or internal secretions. It is interesting to discover that the secretions of the stomach are governed by influences other than psychic or reflex nervous mechanisms. From the facts before us must conclude that gastric secretion may be stimulated by psychic influence, by direct stimulus occasioned by food in the stomach, and, finally, by the specific action of some chemical substance formed in the pylorus and acting through the blood upon the remaining portions of the mucous membrane of the stomach.

We still await the application of these physiological findings to explain certain conditions observed clinically. Although many cases of so-called gastrosuccorrhœa are in reality but instances of retention, there remains a group of cases which are exempt from stagnation, but in which, although the stomach is washed out at bedtime, there is found a considerable amount of gastric juice present in the fasting stomach in the morning. Sometimes the secretion is so excessive as to lead to morning vomiting; in contrast is achylia gastrica, in which no secretion whatever can be discovered. Some patients who suffer from hypersecretion suddenly, and so far unaccountably, experience a diminution or suppression of secretion; in other words, a patient who one day is an example of hyperchlorhydria, on the succeeding day or week will show a continued absence of hydrochloric acid free or combined. When gastric secretion is for a long time absent, we describe the condition by the term "achylia gastrica." This affection, in at least a proportion of cases, seems to be unrelated to any morbid change in the structure of the gastric mucosa. In prolonged cases the glandular structure undergoes atrophy, and restoration of secretion becomes impossible.

It would be easy to explain these cases from the standpoint of inflammatory or degenerative processes in the mucous membrane of the stomach, but such conditions are apparently absent in the early stages of a large proportion of cases. At times the disease is susceptible of satisfactory improvement following local stimulation and general treatment; but a large number remains in which the set of the disease is toward permanent loss of function. Month by month the secretion becomes less, until finally it disappears altogether, often without marked subjective symptoms of gastric disturbance. Lacking the stimulus of the free hydrochloric acid, the pylorus does not contract firmly, and the unchanged gastric contents pass into the duodenum, sometimes very soon after the meal is eaten. In such cases the stomach will be found empty an hour after the ingestion of food, or if the stomach tube is employed soon enough to regain the stomach content, it will be found to have undergone no digestive change except such as depends upon the action of the salivary ferments. Since we have come to look upon the quantity and quality of gastric juice as the normal stimulus of duodenal digestion and as the signal for secretion in the pancreas, liver, and intestine, it is difficult to understand how patients suffering from achylia gastrica are enabled to continue in fair health and with moderately good general nutrition. Such, however, is the case, although it seems to be contrary to what we are led to expect from our physiological study of the relation of the gastric secretion to intestinal digestion. Patients with achylia gastrica followed for fifteen years, while never well, are yet not materially worse than at the beginning of the observations. In some instances there was apparent improvement which coincided with pregnancy and disappeared as soon as the period of lactation had passed.

In health the amount of pancreatic secretion seems to be controlled by the stimulus received in the duodenum from the gastric juice, therefore a very acid secretion should excite a proportionately active duodenal and pancreatic secretion. On the other hand, a lowered gastric acidity should be followed by lowered secretion from the other digestive glands. While this correlation and interaction between the digestive secretions seem normally to proceed in an orderly way, there must occur some deviation from the principle in case of achylia; otherwise we should find the disease to be a much more serious one than it actually is. Starling shows that the secretion from the pancreas is induced through the action upon it of secretin and apparently without the intervention of nerve mechanism. While this seems probable from physiological experiment, it does not perfectly accord with clinical experience.

On examination of the stools of patients with achylia gastrica, although it is common to find that too great a proportion of the food is passed undigested, and although probably a certain amount of it has undergone disintegration through the action of bacteria, still it is evident that the patients digest and assimilate a reasonable proportion of food, as they are able to maintain their weight at a somewhat lower standard, and to develop a fair, although diminished, energy. We have in the recent accounts of the physiology of digestion an explanation of certain of the phenomena observed in achylia gastrica, but we are embarrassed in that they explain too much. In other words, we find that the victims of achylia gastrica suffer far less than should be expected in view of an analysis of the facts.

It would appear that some of the secretions are activated through certain

stimuli with which we are as yet unacquainted, although it would be difficult to explain the comparatively good state of health which many patients enjoy subsequent to closure of the pylorus associated with gastrojejunostomy. Jaworski affirmed that if hydrochloric acid in weak solution was allowed to remain sometime in the stomach in case of achylia gastrica, pepsin eventually appeared. Following this suggestion, Sailer reaches the conclusion that hydrochloric acid as a remedy should be administered in achylia gastrica, but that it should be given before and not after meals, as is usually practised.

It is interesting in this connection to consider the question of natural and artificial inhibition of peptic digestion. There are cases in which, despite high acidity of the gastric content, depending upon free hydrochloric acid, proteolytic digestion does not proceed; something has blocked the digestive process. The explanation of this inhibition has been sought for in various directions. Apparently there are some natural causes of inhibition, perhaps depending upon the stomach itself through the formation there of antibodies. But certain foods and drugs also exercise an inhibitory action on digestion. The albumoses, gelatin and maltose, for instance, have a definite inhibitory effect, while lactose and most carbohydrates have none. In certain instances the concentration of the gastric juice is found to interfere with the digestion of proteids as well as other foods, and when the secretion of the stomach is diluted the digestive process is apparently favored. These facts may account for the curious behavior of the gastric digestion often observed clinically; the total acidity of the stomach content may be high, the amount of free hydrochloric acid may be above the recognized standard, and yet digestion does not proceed. In reviewing the question Sailer concludes that, after attributing to certain foods and drugs the power of inhibiting gastric digestion, and admitting that the element of concentration under special conditions operates to produce a like effect, there remains a group of facts that may be best explained on the hypothesis of the presence of an anti-pepsin such as has been announced by Danielewsky and Weinland. This substance may have a role, not only in protecting the walls of the stomach, but also, in certain conditions, of inhibiting proteolysis in the gastric contents.

As previously stated, the physiological stimulus for the secretion not only of pancreatic juice, but also succus entericus and bile, is free hydrochloric acid, passing with the chyme into the duodenum. When in the absence of gastric secretion the duodenum is deprived of its normal stimulus, what is it that invokes the production of secretin? Granting that this substance is the natural stimulus for secretion of the liver, pancreas, and intestine, it would follow that intestinal digestion would cease if secretin were withheld from activity. In actual practice we observe that with long-continued absence of gastric secretion, a certain proportion of patients show very defective intestinal digestion. In other cases the intestinal digestion seems very satisfactory, and we are forced to conclude that besides the acid secretion of the stomach there must be some other agent that acts to stimulate the production of secretin. Whatever this agent may be, its presence is more manifest in some cases than in others, if we are to explain thereby the variations seen in the intestinal digestion occurring in cases of achylia gastrica.

It is found that oil passing into the duodenum acts to stimulate the production of secretin with an efficacy second only to that of the acid chyme. Probably the fatty foods in a measure replace the function of the gastric

juice in setting the secretion into circulation in cases of *achylia gastrica*. There would seem to be a clinical confirmation of this statement in the successful therapeutic use of oils in relieving the diarrhœa that is sometimes such a troublesome symptom in *achylia gastrica*.

Another and important matter relates to disturbances in metabolism, and more particularly when this involves the question of functional disturbances of the liver. Careful examination of the stools shows that there is a wide variation in the amount of biliary coloring matter discharged in the intestine. The study of the quotidian secretion of the bile, so far as can be judged by that which escapes through a biliary fistula, confirms the belief that at least one function of the liver shows considerable change in activity from time to time. From the evidence we may conclude that these temporary modifications of hepatic function are directly influenced through the nervous system, through psychic impulses, excitement, fatigue, or depression. Drugs have comparatively little effect on the secretion of bile, apparently more upon metabolic processes, but the state of the nervous system seems to be potent in this regard.

The secretion of bile is stimulated by the administration of biliary salts and in a somewhat less degree by the taking of oils. In health the entrance of acid chyme into the duodenum excites the outflow of bile, a result apparently dependent upon the action of hydrochloric acid upon the mucous membrane of the duodenum, thereby leading to the production and absorption of secretin in the blood. It will thus be seen that the mechanism of the secretion of bile seems identical with that of the pancreatic juice. In each case it seems to require the presence of secretin carried through the blood to the organ in question.

11. Infection in Relation to Digestion.—The importance of recognized infection in the etiology of structural diseases of the liver and biliary apparatus leads to the suspicion that unknown infections of moderate activity have much to do with the variations of functional power of the liver that are commonly ascribed to other causes. It must be owing to an acquired or inherent resistance that the liver is not more markedly and frequently deranged than it is by the occurrence of a relatively unwholesome condition of the gastro-intestinal tract. Clinical experience leads to the conclusion that the constant presence in the intestine of pathogenic microorganisms and other sources of intoxication, which may not materially disorder the economy, must be tolerated by means of some form of immunity. It is further observed that with unusual nervous depression this protective barrier of immunity becomes insufficient; the well-known symptoms of toxæmia result, and not infrequently of infection as well.

Undoubtedly these manifestations are not confined to the liver, for we perceive similar occurrences in the parenchyma of the stomach and intestine in gastro-enteritis. It can scarcely be an error to infer that organs that are so intimately correlated and so largely influenced by the sympathetic system of nerves must react to untoward nerve influences; and under such circumstances, when, so to speak, "the guard is down," the infection most readily breaks through and intoxicants still further perturb the function. We may find illustrative examples most often in young children, as in the effect of fright, injury, or heat exposure.

12. Visceral Blood Supply in Relation to Digestion.—This resistance to disease on the part of the liver seems to be proportioned in a measure

to the liberality of the blood supply, and fails when, from narrowing of the hepatic artery, the supply of oxygen is decreased.

Years ago it was suggested that in case of unusually large and numerous inosculations of the branches of the portal vein with the systemic veins, a sufficient auto-intoxication to produce definite symptoms might result. The explanation of coma in hepatic cirrhosis was adduced as an illustration. The introduction of epiploexy, practised by Thalma, has enabled us to observe effects of such inosculations artificially produced, and it must be admitted that some justification is found for the claim that toxic effects may be induced in this way. Especially is this true before the organism has accommodated itself to the change in the circulation, and presumably, also, when the portal blood is for any reason in a specially toxic condition.

While it is impossible to measure the precise responsibility of each fact that operates in the development of the morbid process, it is evident that weight must be given not only to that which circulates in the blood, but to the volume of blood as well; and it is for this reason that of late more thought has been given to the question of sclerosis in the abdominal aorta and its branches.

Not alone the liver may be the victim of these vascular defects, but also the other abdominal viscera. Thus some forms of pancreatitis are to be explained, and not a few instances of that disease of multiple etiology, colica mucosa. Arteriosclerosis, aortitis, and para-aortitis abdominis may be held to account for many degenerations, functional disturbances, and special symptoms of the digestive apparatus. Among the latter are included neuralgias, motor disturbances and lowered secretion.

In those, free from structural disease of the vessels, troublesome visceral disturbances resulting from vasomotor excitation or depression may occur. Perhaps certain affections whose origin is attributed to perturbation of the nervous system, and which are called functional, are so in a different sense than that conceived. They may properly be charged to morbid conditions of the vasomotor centres.

This would seem to be true especially of mucous colitis, an affection that has been accounted for by many morbid processes, some of them opposite in nature, but all perhaps possessing in common the quality of excitement or depression of the blood supply through disturbance of vasomotor centres or through more direct influence, and hence leading to prolonged congestion or ischæmia of the colon. What is true in this part of the intestine may well be so in other parts and other organs. Vasomotor disturbance is an old conception, long used to explain visceral diseases, and although this view has been carried to extremes, it has never been quite dislodged, and undoubtedly, in a proportion of cases, is a source of trouble.

A passive congestion of the abdominal viscera, so frequently met with as a result of cardiac failure or pulmonary obstruction, at once induces a lowered functional activity and later degenerative structural changes. At first there is depression of the specific secretions with an increased activity of the muciparous glands. The motor function of the stomach and intestine becomes hesitating, irregular, and feeble, discomfort or distress is experienced, digestion is delayed and imperfect, while an exuberant growth of bacteria leads to fermentation and increased toxicity of the gastro-intestinal contents. A lowered resistance of the tissues favors the entrance of patho-

genic microorganisms and the setting up of inflammatory changes of a low order which, with the assistance of an increased toxicity, account for the degeneration of the parenchyma and the successive ingrowth of connective tissue to be observed in the digestive organs in all instances of prolonged portal stasis.

13. False Combinations of Secretion.—Disharmony.—The interrelation of the secretions, the enzymes, and activating substances form a complex but interesting field of study. By conjoint action they apparently lead to amazing results, some of them still remaining incomprehensible. There are both clinical and experimental reasons for believing that disharmony in their physiological reactions may result in profound disorder of the digestive organs and even in the most serious organic diseases. Perhaps no better instance of the latter can be pointed out than pancreatitis induced by experimental injection of the duct of Wirsung with bile, as shown by Opie, or with artificial gastric juice, as in Flexner's experiments. Undoubtedly certain cases of pancreatitis follow obstruction in the ductus choledochus at the ampulla and the escape of bile into the pancreatic duct. Possibly the more acute cases may ensue from the entrance of gastric juice into the pancreatic channels passing from the duodenum. This seems more probable in light of the work of H. U. Williams. He shows that gall-stones may so far dilate the outlet of the ductus choledochus as to make easy the entrance of duodenal contents, including bacteria and chemical agents, which might thus find their way into the pancreas. He succeeded in demonstrating this by experimental work on animals. These dramatic and antagonistic results of misplaced physiological secretions seem in part to be demonstrated by the relief observed to follow the drainage of the biliary tract in chronic pancreatitis. There are doubtless other instances of injury inflicted inside the redouts and not by the enemy. Some of these we rather dimly perceive, others are undiscovered, yet we see their results.

14. The Place of the Biliary Tract in Digestive Pathology.—The gall-bladder is a prolific source of disease in the abdominal organs. The observations of Riedel and the experiments of Mignon show that a great proportion of diseases of the gall-bladder develop as the result of infection, a statement which applies with equal force to the various forms of cholangitis. Without entering into the discussion of the means by which microorganisms find entrance into the bile passages, we find them present there, and as a result, an inflammation of greater or less severity is often established. The more acute infections give rise to suppuration or necrosis of the gall-bladder, while the milder ones set up an inflammatory oedema of the lining mucosa, and thus embarrass the natural ebb and flow of bile. The lining of the gall-bladder is not destroyed, but is irritated, and, as a result, an excess of cholesterin is secreted and the changed condition of the bile contained in the gall-bladder leads to the formation of calculi, in the centre of which, if the stones are recently formed, colonies of bacteria are usually found. The conditions which seem most favorable to the enlargement of a biliary calculus appear to be a slight inflammation of the lining of the gall-bladder with temporary and recurring obstruction to the cystic duct. Under these conditions the stone is likely to increase by laminated formation, and its presence in an irritated gall-bladder may excite spasm of its walls, which induces the symptoms known as hepatic colic. Sometimes a calculus is extruded; occasionally lodging at the outlet of the cystic duct, at other

times passing into the ampulla, finding its way through the papilla, it escapes into the intestine.

The inflammation of the biliary apparatus and the increased tension put upon it lead to more or less permanent changes in the parts, such as dilatation of the biliary canals, widening of the outlet of the choledochus, and inflammatory adhesions uniting the gall-bladder to the surrounding parts. Concurrent with these events there is usually much sympathetic disturbance of other viscera, especially the abdominal, sometimes of a character so intense as to distract the attention of the patient and physician from the original seat of trouble. With the biliary apparatus crippled as above described, future infection is facilitated, and recurrence of cholecystitis and cholangitis are frequent unless the patient acquires an immunity through improvement in the general health.

15. Some Peculiarities of Pancreatitis.—As before stated there seems to be a definite relation between diseases of the biliary apparatus and pancreatitis. There is accordingly a belief that this disease, whether acute or chronic, follows upon the entrance of infectious microorganisms through the duct of *Wirsung*. While this is doubtless true in a proportion of cases, there are yet instances of acute hemorrhagic pancreatitis in which no infection was demonstrable, despite careful search. It is recognized that the destruction of the gland and the production of the fat necrosis of *Balser*, so often an accompaniment of the acute forms of the pancreatitis, are definite results of autolysis of the gland structure through the action of its own ferments and the digestive effect of these ferments on the fatty tissues in various parts of the abdomen, and even (through the lymphatics) in other parts of the body. Hemorrhage into the substance of the pancreas occurs at times without trauma, inflammation, or other assignable cause. It remains one of the unsolved questions.

16. Possible Correlation of the Abdominal Viscera through the Sympathetic.—There apparently exists a relation between the suprarenal glands and the functional activity of the pancreas and liver. The appearance of glycosuria in instances of intoxication with adrenal extract seems to bear on this relation. The abdominal sympathetics are associated closely with the function of the suprarenals as well as that of the pancreas and liver.

Meltzer found that when the cervical sympathetic ganglion was removed from animals their pupils dilated widely upon the instillation of adrenalin solution. *Loewi*, suspecting that inhibition of sympathetic nerve influence might lead, through action upon the function of the liver, to hyperglycæmia and glycosuria, and supposing this sympathetic inhibition to be connected with the pancreas, practised the ablation of this organ. In the animals thus operated on there appeared the usual glycosuria, and, in verification of his hypothesis, when adrenalin solution was instilled in the animals' eyes marked mydriasis immediately appeared. Following this he applied adrenal solution to the eyes of 18 diabetic patients, and of these, 10 at once showed mydriasis. In 28 non-diabetic patients the reaction failed in all but 2. One of these proved to have obstruction of the duct of *Wirsung*, and the other had *Graves' disease*.

These results indicate that the functions of the pancreas, the liver, and the adrenal bodies are related to the sympathetic nervous system, and the same is probably true of the kidney.

Sialorrhœa, as a definite symptom of pancreatic disease, is largely discredited, yet its concurrence must be admitted, sometimes in pancreatic calculus, again in cancer of the head, and occasionally associated with transient glycosuria. The reality of so-called pancreatic diarrhœa should not be too quickly abandoned. By the study of the unusual phenomena we may dispel the mist that obscures the field wherein plays some of the most incomprehensible vital processes.

17. Splachnoptosis and Overtension of the Peritoneum.—The displacements of viscera contribute in several ways to derangements of digestion. Gastropptosis may interfere with the motor function of the stomach through increased traction upon its membranous supports, thus giving rise to discomfort as well as interference with the blood supply. The results of displacement of the small intestine are less understood, but ptosis of the colon, including displacement of the cæcum and the sigmoid, is not infrequently the source of symptoms and of disease. The shortening of the ascending colon, thus raising the cæcum to the upper part of the abdomen, the exaggeration of the flexures, and the displacement of the transverse colon downward so that it appears as a long loop, occasionally as a double loop reaching the pelvis, are among the dislocations. Jacobi pointed out that the sigmoid flexure in the fetus is relatively very long, and this disproportion occasionally continues through infancy, even to adult life. Another common defect is the extension of the so-called omega loop of the sigmoid to the right until it may reach the region of the cæcum. This is occasionally associated with extremely sharp bending of the sigmoid. The displacements of the stomach and the colon are apparently more important than those of the small intestine; at any rate, they are better understood. From excessive angulation of the sigmoid, stasis and dilatation of the colon are frequently developed. This angulation is also a contributing factor in exciting sigmoiditis, and is a source of embarrassment in the treatment of that affection. Hernia, whether internal or external, is a serious menace, because of the danger of strangulation. It is not so much appreciated that incomplete hernia is often the cause of more or less continuous abdominal distress and sometimes severe pain which is experienced in regions somewhat remote from the point of irritation. This is seen in the very minute epigastric herniæ which frequently are found in the linea alba, and also in incomplete and unsuspected herniæ of the inguinal and femoral canals. As these symptoms have been repeatedly shown to result from external hernia, it is fair to presume that like results obtain from internal hernia. It is probable that some of the patients who have undergone repeated abdominal explorations, hoping for a discovery of the source of the pain and other symptoms, are in fact suffering from unsuspected traction of some part of the peritoneum or intestine which is caused by hernia.

As vestiges of past inflammation, adhesions of one part of the peritoneum with another sometimes remain, and the unnatural traction thus induced may so far irritate the peritoneum as to occasion pain and, reflexly, considerable derangement of the digestive processes; or there may be sufficient adhesion of the intestine or stomach to embarrass peristaltic movement and delay the onward passage of the contents. The symptoms thus induced, while of little moment in the more phlegmatic, become important in those hypersensitive beings who are prone to have abnormal abdominal sensations, and in whom an harmonious physiological digestion is more often the exception

than the rule. In such individuals relief is sometimes obtained by judicious medical gymnastics and psychotherapy, while surgical measures rather augment the complaint, either because of the new adhesions formed or because the attention of the introspective patient becomes more firmly fixed on himself.

According to Wilms, the pain occasioned by disease or irritation in the abdominal viscera is really produced by means of traction communicated to the mesentery. The cerebrospinal system of nerves, distributed to the parietal peritoneum, is the seat of most abdominal pain. Some think that the splanchnic and pneumogastric nerves are not implicated, but that abdominal pain is excited only in the endings of the cerebrospinal nerves, distributed to the parietal peritoneum (Lennander). Traction upon the mesentery or upon the adhesions irritate these sensory nerves. Overdistention of a loop of intestine leads to stretching of the mesentery and indirectly to tension of the parietal peritoneum.

From this point of view it is easy to understand how pain may be excited in regions somewhat remote from the seat of mechanical pressure or tugging, such as occurs in hernia, adhesions, and visceral displacements.

18. Psychoneuroses in Relation to Digestion.—Frequently an association exists between digestive disturbances and the more definite neuroses and psychoses. Long continuance of infection of the digestive tract inducing a lowering of the general nutrition, leads to depression or irritation of nerve tissue, and sometimes appears to be the exciting cause of brain disease and also adds to its continuance. This is shown by the marked improvement that follows an enlightened course of dietetic and other treatment especially directed to the betterment of primary digestion. On the other hand, the lowered vitality, induced by definite nerve disease, is almost invariably accompanied by digestive disturbance. Illustrations of this fact are observed when the nervous disease rests upon inheritance rather than upon long-continued nerve strain. It is probable that infection and toxæmia are much concerned in these morbid processes, both nervous and mental. With a lowered resisting power, infection of the digestive tract is easily excited.

Under these conditions the auto-intoxications increase because of accompanying faulty elimination. It is commonly observed that victims of nervous or mental disease suffer from urinary inadequacy, and from depression of the other emunctories. To this may be added the element of faulty metabolism which succeeds upon disturbance of organs of internal secretions, as, for instance, the adrenals and thyroid. Enteroptosis with atony and dilatation of the hollow abdominal viscera is frequently encountered in the neurotic and the insane. Besides, congestion and degeneration of the glandular structures often occur, including the liver and pancreas, as well as the secreting tissues of the stomach and intestine.

19. Variations in the Intestinal Flora in their Relation to Diseases of the Digestive Tract.—The character of the intestinal flora, particularly that of the colon, is a matter requiring careful consideration. The importance of this subject is emphasized by the suggestions of Metchnikoff and the results of treatment reported by Tissier. When patients are deprived of foods containing animal proteids, remarkable improvement is reported to follow the deliberate attempts to plant new orders of bacteria, the presence of which is antagonistic to those microorganisms more especially concerned

in the putrefaction of animal proteids and the incidental production of toxic substances.

To recur to the question of motor insufficiency and imperfect drainage, the importance of which, in the clinical course of digestive disease, has been pointed out, the matter appears more conspicuous in the light of the bacteriology of the intestine.

Indication for attention to this source of trouble may be found in the urine. While we know that certain urinary toxins are the products of metabolism more or less normal, and hence depend upon the state of the cellular tissues, clinical observation and laboratory study reveal that, to a considerable extent, other urinary toxins develop in the intestine.

The infections of the gastro-intestinal tract occur both with and without inflammation, but in either event they are accompanied by derangements of secretion. Subacute gastro-enteritis may be followed by degeneration of the glandular parenchyma and even by exudation into the muscular layers, followed by round-cell infiltration and subsequent motor weakness of the stomach and intestine.

It is because of the similarity in the symptoms between functional derangements on the one hand and of infection and inflammation on the other that there has arisen the confusion in differentiating these conditions. The difficulty is not lessened by the fact that infection is prone to follow certain of the neuroses; in fact, they often co-exist. Lowered vitality of a part favors infection, and then there exists a causal relation between neurasthenia and appendicitis, colitis, or other inflammatory involvement of the digestive apparatus.

CHAPTER II.

DISEASES OF THE MOUTH AND SALIVARY GLANDS.

By DAVID RIESMAN, M.D.

A THOROUGH examination of the mouth by all available methods is of great value, not only in the study of diseases localized in or confined to the oral cavity, but also in the investigation of general morbid states. Unfortunately, the present age trusts so much to its instruments of precision that the powers of observation, the training of which made our predecessors diagnosticians of no mean calibre, are permitted to lie dormant. But there is no reason why dexterity in the use of scientific instruments should not go hand in hand with skill in the use of the unaided senses.

A proper study of the mouth comprises inspection of the lips, gums, teeth, tongue, floor of the mouth, salivary glands, and general buccal mucous membrane. In cases of disturbed taste, false teeth and plates should be removed to ascertain their condition and that of the parts with which they are in contact. The sensation of taste should be tested in the accustomed ways; the saliva should be examined as to its consistency, quantity, odor, reaction, and chemical composition. A bacteriological study of the mouth may at times prove useful, and should be undertaken when gonorrhœa, diphtheria, tuberculosis, or other specific process is suspected. Palpation may often aid inspection.

Bacteriology.—The mouth is the constant habitat of many bacteria, by far the largest number of which are harmless saprophytes; but there are some that possess pathogenic properties, which they display, either in the mouth or elsewhere, when suitable conditions obtain. If the care of the mouth is neglected, the number and variety of pathogenic and non-pathogenic bacteria increase. The bacterial flora of the mouth is of course derived chiefly from without, from the air, food, and drink; but when infective processes are active in the organs or parts communicating directly or indirectly with the mouth, the bacteria concerned in them may find their way to the oral cavity. Thus in pulmonary tuberculosis, tubercle bacilli may be deposited by the sputum in the mouth; and it has been shown by Riedel and Kraus that the salivary glands are capable of eliminating bacteria circulating in the blood. Such bacteria may then appear in the saliva.

Despite the fact that the oral cavity teems with bacteria, wounds about the mouth usually heal by first intention. This is to be explained by the fact that the saliva is after all, as Clairmont¹ has shown, not a good culture medium for bacteria, although its bactericidal power is extremely weak.

Aside from individual differences, there is a natural difference between the saliva of the submaxillary and that of the parotid gland. In most animals

¹ *Wien. klin. Woch.*, 1896, Nr. 47.

the first furnishes a secretion that has only a slightly deleterious action upon bacteria; while parotid saliva is capable of inhibiting the growth of several species of organisms. Those of the pyogenic group seem to suffer most in that respect, especially if the secretion is artificially stimulated. When abundantly secreted, the saliva may become sterile.

The researches of W. D. Miller¹ have shown that there is a group of bacteria more or less peculiar to the mouth. Their identification, however, is not yet complete, as the majority have been found in stained preparations only, and have not been cultivated on artificial media.

The following are the most constant mouth bacteria:

Leptothrix innominata (Miller) occurs in all mouths where a white deposit is found along the gum margin.

Bacillus maximus (Miller) is the largest of the buccal organisms and is found especially in dirty mouths. It is similar to the *Leptothrix buccalis maxima* (Miller), the difference being that the latter does not give the iodine reaction, and has segments of greater length; but it may be that the two are identical.

Iodococcus vaginatus (Miller), according to Migula,² is really a mixture of bacteria.

Micrococcus gingivæ (Miller), found in pyorrhœa alveolaris and also in healthy mouths, produces abscesses in mice.

Cladothrix buccalis is found attached to the teeth and in carious dentine.

Streptococcus brevis is the most common species of mouth bacteria. By using it as a test, Gordon was able to detect contamination with minimal quantities of saliva, and to prove that during speaking, particles of saliva have been dispersed a distance of forty feet in front and twelve feet behind him.

Bacillus gangrenæ pulpæ is found almost constantly (95.3 per cent.) in diseased pulps and in dental caries. It possesses the power of producing gangrene of the pulp and softening of the teeth.

Bacillus necrodentalis occurs in deep layers of carious dentine.

Bacterium gingivitis (Kruse) was found in the mouth during an epidemic of scurvy.

The other bacteria that have been found in the mouth, and which are more important from a clinical point of view, are *Staphylococcus pyogenes*, *Streptococcus pyogenes*, *Bacillus pyocyaneus*, and the pneumococcus. These are sometimes found in the mouths of healthy persons. Longcope and Fox³ have shown that virulent forms of the streptococcus and pneumococcus are more common in the mouths of healthy persons during the winter months than at other times.

Friedlaender's pneumobacillus, *Bacillus pseudodiphthericus*, and *Bacillus diphtheriæ* have also been found. The last is of course present when diphtheria exists; but may also be present in a considerable number of healthy children. Erich Müller⁴ found it in 24 out of 100 children examined.

The *Bacillus tuberculosis* is present in the fluids of the mouth in persons suffering from laryngeal and pulmonary tuberculosis, when the sputum

¹ *Die Mikroorganismen der Mundhöhle*, Leipzig, 1892.

² *System d. Bakterien*, ii, p. 218.

³ *Bulletin of the Ayer Clinical Laboratory, Pennsylvania Hospital*, iii, 1906.

⁴ *Jahrbuch f. Kinderheilk.*, Band xliii, 54.

contains the organism. This makes the saliva a means of disseminating tuberculosis.

The bacillus of glanders, the bacillus of rhinoscleroma, and *Bacillus lepræ* may be found in the mouth.

The *Bacillus fusiformis* of Vincent, according to its discoverer, is normally present in the mouth.¹ Its relation to ulcerative stomatitis is discussed farther on.

The Organism of Foot and Mouth Disease.—Siegel² found in human beings and in cattle suffering from foot and mouth disease a short non-motile bacillus, Gram negative, growing at room temperature on all media, and pathogenic for pigeons, pigs, calves, and young cattle, but non-pathogenic for rabbits, guinea-pigs, mice, dogs, and cats.

The *virus of rabies* is present in the saliva of lower animals suffering from rabies. Its nature is unknown.

Spirochætes.—Spirochætes are motile spiral organisms devoid of flagella and possessing an undulating membrane. They are common in the mouth, but have not yet been sufficiently studied. They are often associated with fusiform bacilli; at times also with a curved, sausage-shaped organism that was originally described by Miller as *Spirillum sputigenum*. The *Spirochæta dentium* of Miller, which was found by Koch in the covering of both carious and sound teeth, and by Migula in association with other dental bacteria, requires further identification.

SEPTIC INFECTION OF ORAL ORIGIN.

Oral Sepsis.—Caries of the teeth, alveolar abscess, and various forms of stomatitis, being bacterial processes, may lead to disease at a distance from the mouth in one of two ways: (a) By the absorption of toxins and bacteria into the blood stream; (b) by the swallowing of pus containing toxins and bacteria.

(a) It is not improbable that some cases of cryptogenetic sepsis, ulcerative endocarditis, etc., have their primary source in the mouth.

(b) William Hunter³ considers oral sepsis a factor of great importance in the pathogenesis of pernicious anæmia. The swallowing of pus in dental carionecrosis, in alveolar abscess, and in stomatitis leads to septic gastritis. The writer has looked in vain in a number of cases of pernicious anæmia for sources and signs of oral sepsis, although he has noticed the glossitis to which Hunter calls attention. Yet whatever the results of suppurative and necrotic processes in the mouth may be, Hunter's insistence on the importance of combating them is entirely warranted. The general practitioner, as a rule, pays too little heed to the condition of the teeth. Wherever there are carious cavities, they should be cleansed and filled, if that is possible. Rotten stumps should be pulled out and replaced by artificial teeth. Not only will mastication and, indirectly, digestion be improved thereby, but possible sources of infection that may eventually impair the health are removed.

¹ *Ann. de l'Inst. Pasteur*, 1899, xiii, p. 609.

² *Deutsch. med. Woch.*, 1891, lxix; 1894, 18 und 19; *Arch. f. Laryng.*, 1895.

³ *Pernicious Anæmia*, London, 1901, p. 200 et seq.

Offensive Breath.—The unfortunate possessor of an offensive breath is seldom directly aware of it. Among the causes of the condition are lack of care of the mouth; mouth breathing due to the existence of adenoids, enlarged tonsils, or badly placed teeth; poorly fitting artificial devices; ozena, empyema of the antrum, tonsillitis, diphtheria (in the last the odor is characteristic); local diseases of the mouth, as the various forms of stomatitis, especially the mercurial form; scurvy and caries of the teeth. In most acute febrile diseases the breath is more or less offensive; in typhoid fever it is quite peculiar. An offensive breath more or less characteristic of the disease is, according to Rolleston, found in cirrhosis of the liver. It is described as cadaverous, and resembles the smell of dried and decomposing blood. It is probably due to a failure of the antitoxic function of the liver and the consequent passage into the blood of poisons generated in the alimentary canal. Hence, it is a bad omen.

A sweetish breath is common in chlorosis and in diabetes; the odor is fruity in diabetes with acid intoxication, also in the non-diabetic forms of the acidoses; a urinous odor is found in uræmia; and a peculiar odor difficult to describe characterizes septicæmia. A permanent offensive breath is found in diverticulum of the œsophagus and in certain cases of atony and dilatation of the stomach. The odor in the latter cases is probably disseminated by the expiratory air, the gases producing it being taken up by the blood and eliminated through the lungs. This is also true of some cases of cystitis with ammoniacal decomposition of the urine. An offensive breath is common in carcinoma of the larynx, in some forms of bronchitis, in pulmonary tuberculosis when there are cavities, and in abscess and gangrene of the lung. A particularly repulsive fetor is sometimes due to putrid plugs in the crypts of the tonsils. In hæmoptysis the breath has a somewhat sour odor. There is sometimes an offensive breath in menstrual disorders and after the use of certain drugs, as arsenic, phosphorus, the bromides and iodides. The offensive odor following the abuse of alcohol is more often due to fusel oil and other ingredients than to the alcohol itself.

Treatment.—Most important is the determination of the cause and its removal, if possible. In addition, the use of antiseptic mouth washes is indicated. Miller highly recommends the following:

R _y —Acidi benzoici	gr xc	(6.0)
Ol. menthæ pip.	ʒxv	(1.0)
Tr. eucalypti	ʒj	(30.0)
Alcohol. absol.	ʒiij	(100.0)

DISTURBANCES OF SENSATION.

A. Disturbance of General Sensation.—(a) **Anæsthesia.**—This is usually of hysterical origin, and more often unilateral than bilateral. As a rule, it extends to all qualities of sensation, heat, cold, stereognosis, and taste. In the rare cases not due to hysteria, the lesion involves the superior or inferior maxillary nerve or the tympanic plexus, or is located in the cerebrum, in which case there is complete anæsthesia of one-half of the body (hemianæsthesia).

(b) **Hyperæsthesia and paræsthesia** (formication, pricking, tingling) are usually the accompaniment of local processes, as the various forms of stomatitis; but they may be due to hysteria or to lesions of the central

nervous system. They may, especially paræsthesia, be the precursors of anæsthesia and paralysis.

B. Disturbances of Taste.—Abolition of taste, agusia, may be general or partial, persistent or transitory. Transitory agusia depends on local conditions, being common in dryness of the mouth, particularly when that is due to oral breathing. If partial, it may affect one-half of the tongue or the anterior or posterior portion. In agusia of the anterior third the lingual nerve or the chorda tympani is involved. In glossopharyngeal lesions the agusia affects the posterior portion of the tongue. Centric agusia, that is, agusia due to disturbances in the gustatory centre in the uncinate gyrus, is theoretically conceivable, but has not yet been demonstrated. As has been stated above, agusia may coincide with general anæsthesia of the mucous membrane, such as is found in hysteria. Hyper-agusia is practically seen only in hysteria.

Paragusia, or gustatory paræsthesia, is common in hysterical persons and in the insane; in the latter it is usually an hallucination. Sometimes the epileptic aura takes the form of an abnormal gustatory sensation. It is also observed in some cases of facial palsy and in old cases of otorrhœa; in both of these it affects the half of the tongue on the diseased side. In persons with fever and in those suffering from gastric disturbances there is at times a total perversion of taste, on account of which, tastes agreeable to them in a state of health become insupportable. The writer has had under observation a man suffering from neurasthenia and moderate arteriosclerosis, who had constantly a sweet taste, for which no cause could be found. There was no diabetes and no abnormality in the mouth to explain it.

In the absence of febrile affections and peripheral nerve lesions, the prognosis in paragusia should be extremely guarded, as it may be a forerunner of mental trouble.

THE LIPS.

In a state of health the border of the lips is of a bright red color, which disappears on pressure, but returns instantly. The lips are smooth or very lightly wrinkled transversely to the long axis, and somewhat dry to the touch, except on the internal aspect. There are marked differences in thickness and in the amount of mucous membrane exposed. A moustache on the upper lip causes it to become thick and also a little elongated. A thick lower lip is characteristic of certain races, and is seen pathologically in myxœdematous idiots. The upper lip is thickened in the scrofulous.

The so-called *double lip* is an hypertrophy or enlargement not of the whole lip, but of the labial glands, which causes a projection between the teeth and the red portion of the lip. The appearance produced is as if the affected lip were doubled, hence the names "*lèvre double*" and "*Doppellippe*." According to Eddington,¹ it occurs in young men, and affects especially the upper lip, and, as already stated, is due to an overgrowth of the labial glands. A lip thus deformed is liable to become dry, cracked and ulcerated. The treatment consists in the removal of a strip of mucous membrane, including all the enlarged glands.

A change in the *shape* of the lips may occur as the result of long-continued

¹ *Glasgow Medical Journal*, 1906, lxx, p. 81.

mouth breathing. The lips are pale, flabby, thin, and shortened in a manner preventing their closure.

Fissure of the lips is common, and occurs usually in the centre, but also at the angles.

Changes in the *color* of the lips and of the mucous membrane of the mouth are common and generally of much clinical interest. The lips are blue or cyanotic on exposure to cold, in asphyxia, in pneumonia, in emphysema, in congenital and in some types of acquired heart disease, in mediastinal tumor, in rupture of an aneurism into the superior vena cava, in polycythæmia rubra, in poisoning with coal-tar products, and in some forms of intestinal toxæmia (enterogenic cyanosis). In one of the writer's patients an attack of enterocolitis was always preceded and accompanied by intense blueness of the lips. Pallor of the lips and oral mucous membrane is found in anæmia, hemorrhage, chronic parenchymatous nephritis, and in aortic stenosis. It is not always a positive sign of anæmia, as the blood may be fairly normal and yet the lips be pale. In conditions leading to inspissation of the blood, as in carcinoma of the alimentary tract, the lips may be of a deep-red, almost maroon color. In diabetes there is at times a striking redness of the lips, which the writer has usually found associated with a similar condition of the ears. In jaundice the mucosa on the inner surface of the lips has a yellowish tinge, which becomes more noticeable if the blood is pressed out with a glass slide. This is a valuable means of detecting jaundice in the colored race. Semmola and Geoffreddi¹ state that if a jaundiced patient forces his mouth wide open, two yellow streaks can be seen on the soft and a portion of the hard palate. In lead poisoning, in addition to the characteristic blue line along the gums, there may be flame-like, bluish patches on the inside of the lips. In the so-called blue-gummed negro, bluish or brownish pigment stains are seen on the gums, rarely on the inside of the lips. The presence of brownish patches on the inside of the cheeks, on the palate, or lips is an important diagnostic sign in Addison's disease.

Unnatural *dryness* of the lips occurs in fevers and in ordinary colds. In the former the lips and teeth are often covered with brownish crusts called *sordes*, which when picked from the lips leave a bleeding surface. *Tremor* of the lip is seen in general paralysis of the insane, in alcoholism, in hysteria, and in typhoid and other long-continued fevers.

Eruptions.—The most frequent is the fever blister—*herpes labialis*, cold sore—usually found on the skin surface and red border. There may be but a single vesicle, more often there are several, which become confluent with the formation of reddish-brown crusts. According to Schamberg,² herpes simplex is most common in childhood and early life; 55 per cent. of the cases occurring between the ages of ten and thirty years. Herpes is an accompaniment of croupous pneumonia, malaria, and cerebrospinal meningitis. It is comparatively rare, in the writer's experience, in influenza, and exceedingly uncommon in typhoid fever. There is a belief among clinicians that the occurrence of herpes in pneumonia is a favorable omen; this is also true of herpes in malaria.

Herpes is of infectious origin; but the cause is probably not a single specific microorganism.

¹ *Twentieth Century Practice*, ix, p. 477.

² *Journal of American Medical Association*, March 2, 1907.

PLATE I



The Blue Line of Lead Poisoning, with a Deposit of Lead in the Lower Lip.

Treatment.—Spirits of camphor freely applied in the earliest stages may abort an outbreak of herpes; the lesions when present may be painted over with tincture of benzoin—several applications are made and allowed to dry; they are repeated two or three times daily. When crusts have formed, ointments, such as cold cream, camphor ice, zinc ointment, etc., favor their early separation.

Inflammation of the Labial Glands.—**Baelz's Disease.**¹—This is a chronic disease affecting the mucous glands of the lips. It begins as an indolent swelling and gradually leads to ulceration, without causing either enlargement of the lymph glands or constitutional symptoms. It probably depends on a special infection, but is not syphilitic, although its appearance may lead to the suspicion that the patient has a labial chancre. There is usually an accompanying superficial catarrhal stomatitis. The disease yields readily to applications of dilute tincture of iodine.

Tumors of the Lips.—The principal tumors of the lips are angioma (hemangioma), lymphangioma, and carcinoma.

Harelip.—This is a congenital deformity, consisting of one or more fissures in the upper lip, resulting from an arrest of development. The fissure is not in the median line, but corresponds to the line of junction between the intermaxillary and superior maxillary bones. Harelip may be single or double. As it is a surgical condition, it need not be further discussed here.

THE MOUTH.

INFLAMMATION OF THE MOUTH.

Catarrhal Stomatitis.—Catarrhal, simple, or acute stomatitis is a mild disease, characterized by redness and swelling of the mucous membrane and by increased secretion of saliva. It is produced by irritants acting not too violently, these irritants being mechanical, thermic, or chemical; as food too hot, too cold, too highly seasoned, or too sour; alcohol, tobacco, sharp edges of decayed teeth, caries of the teeth, tartar, dentition, difficult or fruitless sucking, harelip, the use of pacifiers, etc., improper care of the mouth, and mouth breathing. Catarrhal stomatitis is present in many infectious diseases, partly as a result of the same cause, partly because of decomposition of food in the mouth, lack of care, etc. Thus it is found in scarlet fever, measles, typhoid fever, influenza, smallpox, and gastric and intestinal disorders. It is also present, either alone or as an accompaniment of other forms of stomatitis, after the use of certain drugs, such as mercury, iodides, lead, bromides, bismuth, and arsenic.

Pathology.—Hyperæmia, increased secretion, and proliferation of cells are the changes usually found. In the saliva the ordinary mouth bacteria are present in augmented numbers.

Symptoms.—There is moderate pain, which is increased on the ingestion and mastication of food; infants and young children are apt to cry and to protrude the tongue; sucking is difficult. The saliva is increased in quantity and becomes sticky or pasty. The sense of taste is impaired; the

¹ Unna, *Monats. f. prakt. Dermat.*, 1890, xi, p. 317.

appetite diminishes; and in children there may be fever, and at times even emaciation.

Examination of the mouth reveals swelling and redness of the mucosa; the tongue is tooth-marked; the interdental processes of the gums are enlarged, red, and thickened. The mucosa is covered with a tough, whitish saliva, usually in patches. In severe cases the papillæ of the tongue are prominent and bloody at their tips (papillitis). The breath is offensive.

Treatment.—Aside from the removal of the cause, cleanliness is usually all that is required. Simple antiseptic mouth washes, such as a 2 to 3 per cent. solution of boric acid, sodium borate (2 to 3 per cent.), or potassium chlorate (1 to 3 per cent.) will suffice. If the gums are spongy they may be touched with glycerite of tannin (1 to 10), and a little tincture of myrrh may be added to the mouth wash. In infants, the mouth, after nursing, should be washed with sterile water and the antiseptic then applied on a pledget of cotton or linen.

Aphthous Stomatitis.—Aphthous stomatitis is characterized by the formation of cream-white patches or plaques firmly adherent to and embedded in the mucosa. It is most common in children (97.2 per cent., according to A. Levy¹), but may occur in adults, especially in women at the menstrual periods, after parturition, and during lactation. It is rare before dentition, and increases in frequency with its beginning and up to the end of the second year. There is in some cases a marked tendency to recurrence.

Etiology.—The bacteriology has not been thoroughly studied; but Levy, in eight cases, found staphylococci associated in one with *Oidium albicans*. Streptococci were not found. Faulty hygiene of the mouth, decomposition of food remnants, and the irritative influences of dentition are predisposing factors. Whether the disease is contagious or not has not been definitely established; not infrequently it occurs in several members of the same household.

Symptoms.—Aphthæ are small, round, or oval plaques or ulcers varying in size from that of a pinhead to 0.5 cm. in diameter. They are sharply circumscribed, and surrounded by a reddish, slightly elevated border. The favorite sites are the tip and edges and under surface of the tongue, the inside of the lips, and the labiogingival pouch. Their number is usually small, although at times new ones appear as the old ones undergo healing. Often two ulcers are located opposite each other on the gums and on the lip. The pain is usually intense, quite out of proportion to the importance of the affection, and interferes with mastication. Salivation is present, the tongue is coated, the breath offensive, the temper irritable; there may be a little fever. Reference has been made to recurrent aphthæ; in persons predisposed to them any trivial disorder of the general health may bring on an attack of aphthous stomatitis.

Treatment.—This consists in the use of antiseptic washes and, if necessary, mechanical cleansing of the teeth by a dentist. The ulcers may be touched with caustic, preferably the solid stick of silver nitrate. Baginsky recommends the application of a solution of potassium permanganate (1 to 150). Hirsch advises tincture of iodine or a 10 per cent. solution of chromic acid. Alum is also useful.

¹ *Wien. med. Blätter*, 1897, xx, pp. 39, 59, 76.

A *confluent form* of aphthæ in children is described by du Pasquier.¹ It is a serious affection, and is either primary or secondary to bronchopneumonia, gastro-intestinal disorders, or one of the exanthems. The lesions may appear benign at first, but are rebellious to treatment. There is fever and emaciation; at times an infectious erythema appears and constitutes a grave symptom. The disease lasts from one to two weeks, and may terminate in bronchopneumonia or meningitis. There is also a fulminant form ending fatally in twenty-four or forty-eight hours after the appearance of the aphthæ. The most striking postmortem finding is a fatty degeneration of the liver.

Bacteriological examination of the oral lesions shows a polymicrobism, various round and rod-shaped organisms and Vincent's spirilla being found. It is quite probable that this form of stomatitis and the ordinary aphthous form are two etiologically different diseases.

Ulcerative Stomatitis.—*Synonyms:* Stomacace; phlegmonous stomatitis; putrid sore mouth. The term ulcerative stomatitis is loosely used to designate ulcerative conditions in general as well as a specific disease of indeterminate etiology. It occurs in children and adults, and is largely due to lack of care of the mouth, decayed and sharp-edged teeth, and in children to improper feeding. Crowding of the teeth is also an important factor, ulcers not rarely forming on the cheeks and gums from pressure by wisdom teeth. Poisoning with mercury, lead, phosphorus, and iodine is a predisposing cause, as are also infectious diseases, scurvy, etc. Older writers have described epidemic outbreaks of ulcerative stomatitis, especially in barracks; but the condition was probably a different one, in some cases perhaps foot and mouth disease.

Bacteriology.—In addition to the common pyogenic organisms, Bernheim² and Nicolot and Marotte³ found the fusiform bacillus of Vincent, which probably is an important factor in the production of the disease. This bacillus is a polymorphic anaërobe, growing at 37° C., on serum-containing media. It is practically always associated with spirochætes, and is found in a great variety of affections, *e. g.*, ulceromembranous angina, hospital gangrene, tropical ulcer, mercurial stomatitis, scurvy, pulmonary gangrene, peribuccal abscesses, and noma.

Symptoms.—The process, which is almost invariably limited to the region of the teeth, begins at the margin of the gums. The gums swell, become dull red in color, and are covered with a veil-like haziness; subsequently they become yellowish and necrotic, bleed easily, and stand away from the teeth. In the early stages the swollen gums may in part envelope the teeth, while the interdental processes protrude as thick red ridges. As the edges ulcerate and melt away, the teeth become more and more exposed. By lifting away the gums from the teeth, ulcers may sometimes be discovered quite early on the dental surface below the free edge. The ulcerative process may spread to contiguous parts of the lips, cheeks, or tongue, and the teeth, freed from their gingival covering, may drop out. Salivation is profuse, and the odor of the breath horribly fetid. Chewing and swallowing are difficult, and in children there is fever; even in adults there may be

¹ *Rev. mens. d. mal. de l'enf.*, 1903, xxi, p. 353.

² *Centralbl. f. Bakt., Paras. u. Inf.*, 1898, xxiii, p. 177.

³ *Rev. de Méd.*, 1901, xxi, p. 317.

slight rises of temperature. The duration varies from a few days to several weeks, but as a rule is not more than ten days.

Treatment.—Removal of the cause is of prime importance. The teeth should have careful attention and the mouth should be cleansed with antiseptic washes, such as the liquor antisepticus or hydrogen peroxide. Internally potassium chlorate in small doses seems to be of much value. Potassium chlorate is toxic, and should be administered with caution, especially in children; its use should not be prolonged. In severe cases the employment of silver nitrate locally is helpful.

A few words may be devoted to *mercurial stomatitis* (salivation), a sub-variety of the ulcerative. Its mode of origin is not altogether clear, but it is generally held that the mercury, by its reducing action, injures the tissues and in that way prepares the way for pathogenic bacteria. Some persons have an idiosyncrasy to mercury, and in them a single small dose, or perhaps two or three small doses, will produce salivation. Preëxisting diseases, such as chronic nephritis, cirrhosis of the liver, etc., favor it. Infants without teeth and the edentate aged are not liable to mercurial stomatitis. In grave cases death may ensue from extensive ulceration or gangrene and septic absorption.

The *treatment* consists in the discontinuance of the mercury and the use of antiseptic washes, as hydrogen dioxide (2 to 3 per cent.), and, internally, potassium chlorate, gr. ij to iv (gm. 0.12 to 0.24). Mikulicz and Kuemmel¹ recommend iodoform paste or strips of iodoform gauze laid in the mouth and Bockhart² potassium chlorate in powder or in the form of a 50 per cent. tooth paste, as suggested by Unna.³

In a remarkable case reported by Menetrier and Bouchaud⁴ intense recurring stomatitis was produced by a single mercurial injection, and was cured after five months by the excision of the focus of injection.

Gangrenous Stomatitis.—*Synonyms:* Noma; cancrum oris; Wasserkrebs (German). This is a comparatively rare form of infection of the mouth. Like hospital gangrene, which in some respects it resembles, it was more frequent in pre-antiseptic days. Its chief feature is a rapidly spreading gangrene involving the cheeks, gums, and alveolar processes. It is more common in childhood and in hospital than in private practice. Although occasionally an independent affection, it is, in the majority of instances, an accompaniment of or sequel to infectious diseases, such as measles, scarlet fever, diphtheria, typhoid fever, whooping-cough, pneumonia, malaria, and dysentery. In rare cases it follows mercurial stomatitis. The old view that it is caused by the administration of mercury has been disproved; as early as 1826 it was denied by Coates.⁵ It usually affects children between the ages of two and five years, and seems to be more common in the spring and autumn; in adults it is exceedingly rare.

Bacteriology.—Various bacteria have been either isolated in culture or found in stained preparations, but for none of them has the crucial test of reproducing the disease by inoculation been done. The most common

¹ *Krankheiten des Mundes.*

² *Monats. f. prakt. Dermat.*, xxxiv, 1902, p. 113.

³ Equal parts of potassium chlorate and a tooth paste compound of calcium carbonate, orris root, soap, and glycerin.

⁴ *Bulletin et Mém. de la Soc. méd. des Hôp. de Paris*, June 28, 1906.

⁵ *North American Medical and Surgical Journal*, 1826, vol. ii.

organisms are the fusiform bacillus and the spirillum of Vincent, which either by themselves or in combination with other bacteria must in the present state of our knowledge be considered the cause of the disease. Streptococci, staphylococci, pseudodiphtheria bacilli, and perhaps true diphtheria bacilli and pneumococci are also found. Hellesen¹ isolated a gram-positive, non-encapsulated diplococcus, which in animals produced a typical and specific necrosis of tissue. Matzenauer² considers noma and hospital gangrene identical, and believes both are due to an anaërobic bacillus.

Symptoms.—The cheek and lip become swollen and the overlying skin glazed, colorless, and hypæsthetic; a small ulcer forms on the mucous surface, rapidly becomes gangrenous, and advances in depth and width until the integument is reached. If the latter also melts down in the gangrenous process, a perforation results. The hole may be small, or the gangrene may speedily involve nearly the entire cheek, the gums, and even the alveolar processes, so that the teeth are exposed and loosened. In the gravest cases, the tongue and palate may become affected. In rare instances, the gangrene involves the outer parts of the cheek and spares the mucosa.

The constitutional symptoms are often strangely mild when contrasted with the local process. Fever may be absent; when present it is usually of a hectic character, at least in the terminal stages. The odor is frightful and indescribable; the submaxillary lymph glands are enlarged. The disease has a very high mortality—from 75 to 80 per cent. The duration is from one to two weeks, death being due to bronchopneumonia or to exhaustion, which in some cases is hastened by a septic diarrhœa.

Noma is nearly always unilateral; recurrences have been noted. Some authors have reported cases of so-called idiopathic gangrene of the mouth, in which the disease set in spontaneously without any preceding illness on the part of the individual attacked.

Treatment.—Removal of the diseased tissue by the knife or Paquelin cautery under anæsthesia, the wound being afterward dressed antiseptically, is the most efficient treatment. Destruction of the odor is imperative, and is best accomplished with formaldehyde, potassium permanganate, or iodoform. Diphtheria antitoxin has been employed in some cases, on the ground that the diphtheria bacillus was present; but it is quite probable that the bacillus was not the Klebs-Loeffler, but the pseudodiphtheria bacillus. Motschan³ has seen good results from the red-light treatment. An abundance of food and alcoholic stimulation are indispensable.

Swoboda⁴ has described a gangrenous inflammation of the *tooth germ* in the newborn, causing a destruction of the gums, with subsequent sequestration of the crowns of the teeth in infants that are still far from the normal period of dentition. The cause is probably a septic infection, and the prognosis is always fatal.

Thrush.—*Synonyms:* Mycotic or hyphomycetic stomatitis; Soor (German); muguet (French). Thrush is a form of stomatitis characterized by the formation of whitish patches largely composed of the causative fungus, the *Oidium albicans*. It occurs chiefly in infants, rarely in adults.

¹ *Jahrb. f. Kinderh.*, 1908, lxvii, 294.

² *Arch. f. Dermat. u. Syphilis*, 1902, 60, p. 373.

³ *Wien. klin. therap. Woch.*, 1904, Nrs. 21 und 22, pp. 575, 606.

⁴ *Die Therapie der Gegenwart*, 1904, xlv, p. 515.

The *Oidium albicans* of Robin, belonging to the class of blastomycetes, is generally accepted as the etiological agent; although some authorities hold the *Saccharomyces albicans buccalis* to be the cause of the disease. The oidium is a pleomorphic organism presenting itself under two forms, small, yeast-like bodies and filaments, both of which are usually found together in the mouth. When cultivated artificially, the reaction and physical properties of the medium seem to influence the form of the parasite, so that with increasing acidity the filamentous form disappears. The virulence of the organism may be augmented by passage through animals. Although in the body the oidium probably secretes a toxin, none has as yet been found in artificial cultures. According to Concetti,¹ if the growth in culture is reduced to a fine powder and centrifugalized, two layers are formed: an upper, composed of the protoplasm and fat (called oidine supérieure, O. S.), and an inferior, containing fragments of membrane and protoplasm (called oidine résiduelle, O. R.). The inoculation of O. R. into animals causes local irritation and death by thrombosis and embolism; O. S. causes no local irritation, but contains most of the protein of the oidium, with its general pathogenic powers. The oidial poison, however, is the result of the action of the oidial protoplasm on *living* cells of the animal body. No immunizing substances result from the action of O. S. or from the soluble products of the fungus. By submitting the fungus to slow desiccation in the presence of caustic potash, an attenuation of its virulence is obtained; and by the use of increasing doses of this substance, an immunization may be effected. The doses must be given intravenously. The immunizing substance is probably contained in the residual oidine (O. R.).

Rajat and Péju² have isolated two types of fungus, one in which the cells are of the average size of yeast cells and the other in which they are from six to eight times as large. The small-celled variety was present in all (70) the benign cases, that is, in those yielding promptly to treatment; the large-celled forms were present in 5 tenacious cases.

Thrush seldom, if ever, appears in healthy individuals; but chiefly in those whose vitality is impaired. In infancy trivial disorders may prepare the way—simple colds, slight intestinal disturbances; while in older children and adults, usually the aged, pneumonia, typhoid fever, and the exanthemata are the predisposing causes. In early life the epithelium is more easily penetrated; moreover, there is in the newborn, according to Grosz,³ a tendency to a desquamative process producing a thin, grayish deposit, which favors the development of thrush. An acid condition of the saliva, which is normal in infants and is easily produced by fermentation of milk remnants, facilitates the growth of the thrush fungus. The too vigorous washing of the mouth practised by some nurses after the birth of the child may act in the same way. The disease is contagious and is transmitted by dirty nipples and dirty feeding bottles.

Symptoms.—The first sign of thrush is the formation of small, whitish points on the tip and edge of the tongue and on the inner surface of the lips, places on which desquamation of the mucous membrane is favored by the act of sucking. The points grow into patches of varying size, and

¹ *Arch. de Méd. des Enf.*, 1900, iii, pp. 449, 517, 590.

² *Comp. Rend. Soc. de Biologie*, 1906, lxi, p. 523.

³ *Jahrb. f. Kinderk.* 1896, xlii, 177

PLATE II

FIG. 1



FIG. 2



Herpetic Inflammation of the Mouth and Tongue.

PLATE III



Herpetic Inflammation of the Mouth and Tongue.

may cover the entire inner surface of the mouth like a coat of plaster. In some cases the disease extends to the œsophagus and stomach; the nose and larynx may also be affected, rarely even the vulva. The patches consist of mycelial threads and epithelial cells, with leukocytes in small numbers. The mucosa is red and bleeds easily if the patches are forcibly detached. Nursing in infants and in older children deglutition and mastication are difficult. There may be slight fever and restlessness; an eczema of the buttocks is sometimes produced. Several writers have recorded cases of thrush metastasis.

Diagnosis.—The diagnosis is easy; all that is necessary is to examine a bit of the deposit under the microscope. Sometimes the patches are mistaken for curds of milk, which in appearance they closely resemble.

Treatment.—For prevention, pasteurization of the milk and cleansing of the bottles and of the mouth after feeding are important. Ordinarily, the disease yields readily to simple measures, such as wiping away the patches with a piece of soft linen or gauze soaked in boric acid or sodium bicarbonate solution, the mucous membrane being afterward brushed with the same solution, or with a weak solution of mercuric chloride (1 to 4000), sodium hyposulphite (1 to 25), or a weak solution of borax or potassium permanganate. Such constitutional treatment as is suggested by the underlying conditions must of course be instituted.

Herpetic Stomatitis; Herpes Buccalis; Stomatopharyngitis Herpetica or Herpetiformis.—Various herpetiform eruptions affect the mouth aside from the simple herpes of the lips already described. Some are associated with herpes of the pharynx; others are limited to the oral cavity, usually its posterior portion. In a case of the writer's, the vesicular eruption involved the tongue, lips, gums, and tonsils, and it was associated with a violent conjunctivitis. (See Plate III.) The vesicles were small, yellowish white, and on the gums became confluent. There was some fever, inability to swallow, salivation, and pain. The condition presented the picture of an acute infection. Only one culture was made, and that was negative. As pneumonia is an occasional, though rare, complication of herpes of the mouth and pharynx, the latter may, like the former, be of pneumococcic origin.

In women an herpetic stomatitis sometimes occurs at the menstrual period.

Treatment.—This consists in the use of the milder antiseptic washes, as, for example, liquor antisepticus (1 part to 2 or 3 parts of water).

Gonorrhœal Stomatitis.—This was first discovered by Rosinski.¹ It occurs in the newborn from five to twelve days after birth, in the form of whitish deposits on the anterior two-thirds of the tongue, leaving a free border from 2 to 5 mm. wide; also on the anterior half arches, and the posterior border of the alveolar process of the upper jaw, along the free edge of the gums anteriorly, and occasionally on the frænum of the tongue and lips. A reddish reaction zone surrounds the exudate. The functional disturbances are slight, and the process heals without scars in from four days to four weeks. In adults it is rare.

The cause in children is infection of the mouth with the gonococcus during birth.

Treatment.—Silver salts in dilute solution (1 to 2 per cent.), as a rule, bring about rapid cure; mercuric chloride (1 to 7000) may also be used.

¹ *Zeit. f. Geburts. u. Gynäk.*, 1891, xxii, p. 216.

Bednar's Aphthæ.—These are small, whitish plaques occurring in the newborn in the region of the hard palate. They are probably due to pressure of the tongue or the nipple during sucking, their development being favored by the general tendency to epithelial desquamation in the first few days of life. Baumm¹ considers the forcible cleansing of the mouth, practised after birth by some nurses, to be the real factor, and strongly advises against it. The little ulcers heal spontaneously as a rule; in rare cases they constitute the atrium of a general infection.

TUMORS OF THE MOUTH.

Carcinoma.—Carcinoma may start in the mucous membrane of the cheeks, the gums, about the angles of the mouth, the lips, or in the floor of the mouth. It is usually of the squamous epithelioma type, more frequent in men than in women, and very malignant. Early operation is the only treatment that promises good results.

Cysts.—The most common cystic tumor of the mouth is the so-called ranula (French *greuouillette*), which may arise from the sublingual gland or its ducts, from Wharton's duct, or from a small group of glands—the *glandula incisiva*—in close proximity to the neck of the middle or lateral incisor. In rare instances a ranular growth may take its origin from the remains of the thyroglossal duct, or, as Morestin² thought in a case observed by him, from the branchial clefts, or finally, from Blandin-Nuhn's glands on the under surface of the tongue, as in a case reported by Foederl.³ It is at times difficult in a given case to determine the origin of a cyst, but it may be remembered that those from the thyroglossal duct have a lining of columnar ciliated epithelium, while those arising from the acini or excretory ducts of the salivary glands have a lining that more or less faithfully reproduces the epithelium of the parent structure.

Suprahyoid and submental cysts are sometimes found associated with cysts of the sublingual region, with which they may communicate, but they may also exist independently.

The ordinary ranula results from obstruction of the gland duct, and is in its nature a chronic tumor. An acute ranula has also been described; it is due to an acute inflammation of the duct of the submaxillary or sublingual gland, or of both.

Cysts of the parotid gland are extremely rare. One with a lining like that of the mouth is reported by Morestin.

Symptoms.—The characteristic symptom of ranula is the presence of a cystic tumor on the floor of the mouth, usually just to one side of the median line, pushing up the mucosa, which is tightly stretched over it, and displacing the various structures of the mouth. Fluctuation is easily obtained.

Treatment.—Evacuation of the contents is an uncertain method, unless combined with strong cauterization of the lining wall. The old-fashioned seton is still in vogue, and may give good results. In some cases excision is necessary, but on account of the bleeding attending the operation, and the difficulty in maintaining anæsthesia, it is not easily performed.

¹ *Berl. klin. Woch.*, 1891, 840.

² *Bull. et. Mem. Soc. anat. de Paris*, 1902, 6 s., lv, 707-711.

³ *Arch. f. klin. Chir.*, 1894-1895, xlix, p. 530.

A *dermoid* on the floor of the mouth has been described by Brentano.¹ *Mixed tumors*, to be more fully described in the section on Salivary Glands, are also to be found in various parts of the mouth.

DERMATOSES OF THE MOUTH.

Various affections of the skin may simultaneously or independently affect the mucous membrane of the mouth.

Urticaria.—In severe cases of urticaria, especially in giant urticaria, similar lesions may appear in the mouth. The tongue and uvula may be enormously enlarged; there is intense burning and thirst and at times alarming shortness of breath. Urticaria is usually toxic in origin, and is particularly likely to follow the eating of shellfish.

Angioneurotic œdema, a condition closely allied to giant urticaria, is characterized by a sudden painless swelling of the parts involved. The lips when affected, and they are a very common seat of the disease, are swollen to an enormous size and stand out as huge, stiff, fleshy masses. It is most common in women, and has a tendency to recur. Gastro-intestinal symptoms, especially nausea and vomiting, may be present, but are not constant. In a recent case of the writer's, in a young married woman aged twenty-six years, it was associated with swelling of the eyelids, and followed gastric derangement.

Treatment.—The œdema tends to disappear spontaneously, and remedies seem to have but little influence over it. In the case cited above, benefit seemed to be gained from calcium lactate, given in 10 grain doses (gm. 0.6) three times a day. If there is any disorder of the gastro-intestinal tract, this must be corrected, and the diet should be simple.

Psoriasis.—It is doubted by many whether psoriasis of the mucous membrane occurs. In the cases where no lesions of the skin existed, the buccal disease was either leukoplakia or some other non-psoriatic condition. A probable genuine case is, however, reported by Oppenheim.²

Lupus Erythematosus.³—This invades the mouth from the face and usually presents itself as superficially inflamed areas, having an elevated, deep-red border marked radially by dilated veins; in the centre of the patches the mucous membrane is atrophic, violaceous, and dotted with countless fine white or bluish-white points or lines. Some of the plaques show erosion of the surface and are covered with a yellowish-white deposit that cannot be detached. In older foci the appearances may be somewhat different—the edges are less elevated, and usually have extending from them fine arborescent (*reisbesenartig*) prolongations. The most frequent seat of lupus erythematosus is the cheek corresponding to the space between the upper and lower teeth. In the differential diagnosis, eczema, lichen planus, pemphigus, leukoplakia, and perhaps psoriasis have to be considered.

Treatment.—This consists in counterirritant and absorptive applications and cauterization, but is usually of little avail. Unlike lupus vulgaris, lupus erythematosus is not greatly influenced by the Röntgen ray.

¹ *Deut. med. Woch.*, 1906, p. 164.

² *Monat. f. prakt. Dermat.*, 1903, xxxvii, 489.

³ See the article of Kren, *Arch. f. Derm. u. Syphilis*, 1907, 83, p. 13.

Lichen Planus.—The lesions of the mouth are nearly always found in association with lichen planus of the skin; in rare instances they antedate the latter. They appear in the form of whitish dots, plaques, or streaks, giving to the mucous membrane an appearance as if it had been cauterized with silver nitrate. The lesions are unattended by any inflammatory reaction of the mucous membrane.

Etiology.—The disease is most common in those whose nervous systems are depressed.

Treatment.—This in the main is general, the common tonics—iron, strychnine, quinine, and cod-liver oil—being the remedies indicated. Arsenic in ascending doses (up to 10 minims of Fowler's solution) is a valuable agent; mercury is also useful, especially in cases in which arsenic fails.

Pemphigus.—Pemphigus may appear on the mucous membrane of the mouth; the lesions being widely scattered over the gums, the roof of the mouth, and the soft palate. Farlow¹ reports two obstinate cases, in which successive crops of vesicles appeared. At various times lesions were also present on the cornea, in the nose, in the vagina, and on the legs. The treatment consists in the use of alkaline antiseptic washes to prevent secondary infection, and the administration of arsenic. Local anæsthesia (cocaine, orthoform) may be necessary to render eating possible.

Acne.—Acne, one of the commonest lesions of the skin, is rarely found on the mucous membranes. Unna² describes a case affecting the upper lip, in a woman who had acne of the mouth and chin. The lesions appeared in the form of hard, yellow, painless nodules, and yielded to treatment with resorcin ointment and ichthyol soap.

Eczema.—Eczema sometimes affects the vermilion of the lips (eczema seborrhoicum, cheilitis exfoliativa).

Tinea Circinata.—This may spread from the face to the lips and mouth.³

Scleroderma.—Scleroderma sometimes attacks the mouth, leading to atrophy of the mucous membrane and to its adhesion to the submucous tissue. It may also cause falling out of the teeth.

Erysipelas.—In rare cases erysipelas of the face extends to the mouth, producing a deep redness and swelling of the mucosa, and profuse salivation. The soft palate and uvula are especially involved, and their swelling causes pain and difficulty in deglutition. In severe cases the tongue may be involved, as well as the pharynx and larynx. The diagnosis is only assured if erysipelas co-exists, or has immediately preceded or immediately followed. In persons having repeated attacks of erysipelas, groups of small vesicles are sometimes found in the mouth. They may be of the nature of lymphatic varices.

CHANGES IN THE MOUTH DUE TO GENERAL DISEASE.

Typhoid Fever.—In addition to the familiar changes in the tongue, typhoid fever produces alterations in the mouth of a less well-known char-

¹ *Boston Medical and Surgical Journal*, 1904, cli, 671.

² *Monats. f. prakt. Dermat.*, 1890, xi, 321.

³ Robinson, *Journal of Cutaneous Diseases*, 1893, 366.

acter to which attention was first called by Bouveret.¹ These changes consist in ulceration, usually on the upper part of the anterior faucial pillar, on the tongue, uvula, cheeks, lips, and labiogingival folds. Devic² observed these ulcerations 81 times in 220 cases of typhoid fever (36.8 per cent.). They appear between the seventh and fourteenth days of the disease, are shallow, and are preceded by a slight discoloration of the mucosa. As a rule they heal in from one to two weeks without leaving any permanent defect. Bacteriologically only the common mouth organisms were found in Devic's cases; but Blume³ discovered the Eberth bacillus in three out of five cases of ulceration of the palate. The regions at which the ulcers occur are more or less points of compression; this, together with the comparative immobilization of the mucosa by reason of the rarity of movements of deglutition, favors infection.

Treatment.—As the ulcers are superficial and heal readily of their own accord, active measures are not warranted; the maintenance of oral cleanliness is sufficient.

Uræmia.—The mouth is frequently affected. There may be an insupportable dryness, or an actual stomatitis which is due to bacterial infection. Instead of dryness of the mouth, there may be salivation without any stomatitis to account for it. Reneon believes that the salivary glands may act vicariously for the kidneys. Uræmia, especially the gastro-intestinal type, may also lead to parotitis.

Diabetes.—The tongue in diabetes is large, red, "beefy," and bordered with a fissured margin; the mouth is dry; the teeth are often carious. Pyorrhœa alveolaris is frequent, and its discovery should always be a stimulus to an examination of the urine for sugar. Diabetes insipidus presents nothing but dryness of the mouth.

Pernicious Anæmia.—In some cases dental carionecrosis and ulcerative processes are found in the mouth, which Hunter considers an important factor in the production of the blood dyscrasia. In the majority of cases of pernicious anæmia seen by the writer such ulcerative conditions were not present. There was only extreme pallor and dryness of the mucosa.

Leukæmia.—The mucous membrane of the mouth may become the seat of lymphoid hyperplasia similar to that found in the lymphatic glands, spleen, and intestines. The infiltrations show a tendency to undergo necrotic change leading to extensive ulceration, and pseudomembranous and gangrenous processes. In acute leukæmia, gingivitis and ulcerative stomatitis may be among the first signs of the disease. As the gums bleed easily, and as there is marked fœtor of the breath, the condition may be mistaken for scurvy. It is always advisable to make a thorough study of the blood in cases presenting stomatitis with hemorrhagic diathesis and a typhoid state.

Scurvy.—The gums in scurvy are pale, bluish, soft, and bleed easily; the teeth are loosened; there is salivation, intense fœtor of the breath, pain, and tenderness. Babes has isolated an organism, *Bacillus gingivitis*, which may be the etiological agent. It is found in association with the streptococcus. Oral changes, it should be remembered, are not always present in scurvy. In the differential diagnosis in a given case, the possibility of acute leukæmia must be kept in mind.

¹ *Ann. des mal. de l'oreille et du larynx*, 1876.

² Troullieur, *Gaz. des hôp.*, February 13, 1908, 18, p. 207.

³ *La Semaine méd.*, January 22, 1908.

Locomotor Ataxia.—Oral conditions found in locomotor ataxia are perforating ulcer (mal perforant buccal) and falling out of the teeth. The latter is usually entirely painless and may be followed by resorption of the alveolar processes. At times there is a painful anæsthesia of the mucous membrane. The buccal changes are probably dependent upon a peripheral neuritis.

Pregnancy.—Hyperæsthesia, paræsthesia, and salivation are common. Sometimes there is inability to chew certain articles of food, which in a case of the writer's, extended only to the eating of celery. Pain in the teeth, for the relief of which the teeth are often foolishly sacrificed, is a common symptom of pregnancy. At times there is a true gingivitis.

Varicella.—Lesions may occur in the mouth in chickenpox, but the vesicular stage is rarely observed.

Variola.—Lesions of the mouth may appear simultaneously with those upon the skin or later; they are in the form of pustules that soon become macerated and transformed into superficial ulcers. The tongue may be spared.

Treatment.—Weak antiseptic solutions of borax or boric acid, potassium chlorate, or potassium permanganate should be employed. The lesions themselves may be touched with dilute tincture of iodine or the tincture of chloride of iron.

Koplik's spots in measles, the strawberry tongue of scarlet fever, and analogous conditions will be found fully described under their respective diseases.

Pyorrhœa Alveolaris.—*Synonyms:* Rigg's disease; periostitis alveolaris dentalis; general periodontitis, etc. This is a widespread affection of obscure etiology, occurring usually in persons over thirty years of age. Involving, as it does, a loss of teeth in the best years of life, it ought to interest physicians much more than it has hitherto done. If physicians and dentists would combine in a study of the disease, perhaps the mystery that surrounds it might be lifted.

Etiology.—Pyorrhœa alveolaris may be classified as local and systemic. The local form is due to the deposition of salivary calculi upon the crown surfaces of the teeth in proximity to the gum and to the pericemental membrane; and the systemic form to vascular disorders in the pericemental membrane, the result of a variety of constitutional disturbances, such as gout, rheumatism, syphilis, tuberculosis, Bright's disease, etc. It would also appear as if heredity were a factor in its production. Pyorrhœa of gouty origin is the constitutional variety most frequently observed, and is of particular interest to the physician, inasmuch as it bears to the alveolo-dental articulation the same relation as the gouty deposits bear to the articulations of the joints.

The course of the disease in both varieties of pyorrhœa is characterized by a gradual destruction of the cellular elements of the pericemental membrane, and by an inflammation of the gums overlying the affected teeth. The inflammatory process is of polymicrobial origin; but bacteriological studies have shown that a principal part is played by the pneumococcus. In the case of systemic pyorrhœa no calcareous deposit is to be seen upon the exposed surface of the tooth; but on pressing the gum a pyogenic exudate will at once appear. In such cases the deposits are located high upon the roots of the teeth concealed from view, and are composed of calcium

salts derived from the blood (hæmatocalcic deposits), or in gouty pyorrhœa, of a nucleus of urates of sodium and calcium enveloped by a layer of calcium phosphate. In the local variety the deposits are found upon the exposed surfaces of the crowns, especially the lingual surfaces of the lower incisors and laterals and the buccal surfaces of the upper second and first molars, and are derived entirely from the saliva (ptyalocalcic deposits).

Symptoms.—The gingival mucous membrane appears congested and is of a deep-red or purplish color. Becoming detached coincidentally with the destruction of the pericemental membrane and the alveolar osseous tissue, pockets are formed between the root and the alveolus, which are almost constantly filled with inflammatory products, food débris, and saliva. On pressure against the gums, these pockets may be emptied of their purulent or semipurulent material. The affected teeth, which are tender to the touch, but usually free from caries, gradually become loosened and eventually exfoliated. A general catarrhal condition of the mouth and a characteristic fetor ex ore are usually present. The disease may persist for months or years, and while it lasts there is constant swallowing of the purulent exudate and tissue débris, which in time may give rise to local disturbances in the stomach and to general conditions that may be regarded in the light of a pneumococcus toxæmia. The possible part played by these factors in the production of pernicious anemia has been noted.

Diagnosis.—But little difficulty attends the diagnosis. As a rule, several teeth are affected, chiefly the upper incisors. The swollen receding gums, and the oozing of pus from pockets beneath the margin are easily discovered signs. It should be borne in mind that pyorrhœa alveolaris may be an early symptom of diabetes mellitus and of tabes dorsalis, and that it may exist for years without being perceived by the patient.

Treatment.—Patients suffering from pyorrhœa alveolaris, local or systemic, should be referred to the dentist for oral treatment. The treatment of the local form is almost entirely mechanical, consisting in the removal of all salivary deposits and the scaling and polishing of the surfaces of the teeth. As a stimulating application to the gums after mechanical cleansing, either tincture of iodine or glycerite of tannin may be used. The treatment of the systemic form is much more intricate and requires the conjoint services of the physician and the dentist. It is the former's province to eradicate whatever constitutional disturbance may be responsible for the oral disease; and that of the dentist to treat the local condition by such means as may be indicated by the state and location of the affected teeth. As a stimulating and antiseptic mouth wash, the following gives satisfactory results:—

R _y —Hydronaphthol	gr. xv	(1.0)
Alcohol	5j	(30.0)
Water	5j	(30.0)

Sig.—Thirty drops in a glass of warm water twice daily as a mouth wash.

TUBERCULOSIS OF THE MOUTH.

Tuberculosis of the mouth is rarely primary, at least not in the pathological sense; clinically, it may be; at any rate, cases occur in which, aside from the oral or salivary tuberculosis, no other tuberculous lesion is demonstrable. At times, however, repeated careful examination may reveal

another focus that constitutes the primary source. In the tonsils an apparent primary localization of tuberculosis has been described.

Tuberculosis in the form of lupus may spread from the face to the mouth. The ordinary form of tuberculosis is usually the result of a direct infection of the mouth with bacteria from the sputum. If there is a hæmatogenic infection, it has not been demonstrated. It is not likely that this occurs in the tongue, as muscle tissue in general is seldom affected by tuberculosis. Tuberculosis of the *lips* is occasionally seen in tuberculous subjects. It assumes the form of an ulcer, which is not easily distinguished from chancre.

Tuberculosis of the Tongue.—The tongue is the commonest seat of oral tuberculosis, although it is rarely affected, which is surprising when one considers how exposed the tongue is to infection from the sputum passing over it. Cases are recorded in which the sputum did not seem to be the cause of infection; but in which the latter came from without. The only instance seen by the writer occurred in a street-car conductor, who had the objectionable but not uncommon habit of wetting the transfer tickets with his tongue, so as to be better able to separate them with his fingers.

The disease appears in one of several forms, as circumscribed submucous nodules (tuberculomas), which in time ulcerate, as a disseminated process, and rarely in the form of tuberculous fissures or rhagades. The nodules are either single or multiple, and may remain stationary for a long time. Eventually, ulcers form which are shallow, irregular in outline, have undermined edges, and are covered with a grayish-white slough; toward the margin miliary nodules may be seen.

Ulcers occurring in association with advanced tuberculosis of the lungs have a greater tendency to spread in extent and depth than those that are apparently primary. The lymph glands are rarely affected. There is not much pain in the usual type of chronic non-spreading or slowly spreading ulcers.

Diagnosis.—A careful history and the discovery of tuberculosis elsewhere aid materially in the diagnosis. In differentiating tuberculous and carcinomatous ulcers, the following points are important: tuberculous ulcers may be multiple; the edges are softer and less infiltrated; there is less pain, and less frequent involvement of the lymph glands than in carcinoma; the age of the patient is also of importance.

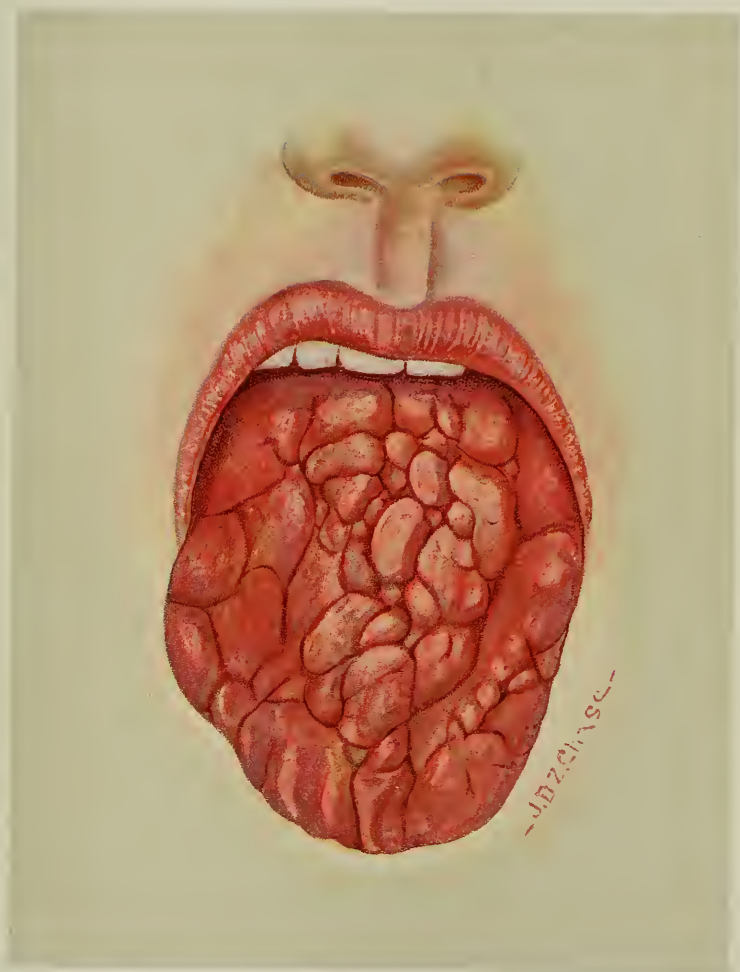
Treatment.—If the pulmonary disease is far advanced, treatment is hardly worth while. In other cases, total excision of the tuberculous area is the best procedure. If not possible, the ulcers may be removed with the thermo-cautery or sharp spoon. For the dressing of the ulcers, iodoform is best.

Tuberculosis of the Gums.—Miliary nodules may appear on the flesh of the gums; ulceration may occur along the free edge and extend to the hard palate. The condition has followed tooth extraction in tuberculous subjects. Tuberculosis of the soft palate sometimes occurs in persons with pulmonary tuberculosis. Two cases of this kind have been reported by Katz.

SYPHILIS OF THE MOUTH.

In the clinical course of syphilis, lesions of the mouth play a prominent part. The *primary sore* is found there in the largest number of cases of so-called extragenital syphilis. The lesion is the counterpart of that found

PLATE IV



Syphilitic Lobulation of the Tongue. "Cobble-stone Tongue."

on the genitalia; but it may be only a fissure or a superficial flat erosion, with a pseudomembranous covering. It is usually single, and, even if small, leads to marked glandular enlargement. Its commonest sites are the lips, especially the lower, the tongue, the hard and soft palates, and the tonsils. The danger of infection to others renders it important to make a correct diagnosis early. If it is a case of syphilis insontium, the patient is generally unaware of the nature of his ailment. In the diagnosis tuberculous and cancerous ulcers have to be considered.

Secondary Lesions.—The secondary lesions found in the mouth are an erythema and the so-called mucous patch (condyloma latum; plaque muqueuse; plaque opaline). The latter, which is among the most infectious of the syphilitic lesions, is a pearly or milk-white area upon a red inflamed surface. The patches are irregular in outline and variable in size and number. They are found in all parts of the mouth, most frequently upon the lips, the palatal arches, and the tonsils. At the angles of the mouth they lead to rhagades. They appear about the same time as, or before, the roseola on the skin and may be painful or painless.

Treatment.—The local treatment consists in the use of antiseptic washes in the mouth and direct application of 1 per cent. mercuric chloride solution, or a solution of nitrate of silver (10 to 20 per cent.), or chromic acid solution (5 to 10 per cent.). It is best to use wooden applicators and wooden tongue depressors, and to burn them afterward.

Tertiary Lesions.—Tertiary lesions in the mouth are of the nature of gummata, and require no special description. Their most frequent sites are the hard and soft palates; but they may be found in the tongue, lips, and rarely in the cheek. In the palate they usually lead to perforation. The tertiary lesions of the tongue may according to Fournier be divided into two types—sclerosing and gummatous glossitis. In the former the infiltration does not break down, but cicatrizes and gives to the tongue a nodular or puckered appearance (langue lobée, langue rhagadiforme). The gummatous lesions break down and ulcerate. A tertiary lesion may be mistaken for a primary sore, but, besides being usually deeper, it is not accompanied by marked adenopathy. The history, moreover, is helpful.

Treatment.—The treatment consists in the vigorous use of mercury and potassium iodide.

Smooth Atrophy of the Tongue.—Smooth atrophy of the tongue is an atrophy of the epithelial covering and of the large sebaceous glands at the base of the tongue, which is found more often in acquired syphilis than in any other condition, and has been considered a pathognomonic sign of this disease. Goldschmidt¹ found it in 17 per cent. of 140 cases of secondary, and in 47 among 60 cases of tertiary syphilis. But as it is also found in non-syphilitic subjects, it cannot have the diagnostic value ascribed to it. In examining for it, the finger or the mirror may be used. With the former, the unnatural smoothness of the base of the tongue is perhaps more readily detected.

Hutchinson's Teeth.—A crescentic notching of the teeth, especially of the upper central incisors, was described by Jonathan Hutchinson, and was by him and others considered a sign of inherited syphilis. It is found in the permanent set, and disappears with advancing years through the wearing

¹ *Berl. klin. Woch.*, 1899, Nr. 43.

down of the edges of the teeth. Syphilis is, however, not its only cause—any nutritive disturbance in early life may lead to notching of the teeth. Thus Roussel¹ reports the case of a woman, aged twenty-three years, who was neither syphilitic nor rachitic, who had a general ichthyosis and Hutchinson's teeth. Seldom, however, is the regular crescent found in conditions other than syphilis, and the presence of such teeth always suggests a careful search for additional evidence of congenital lues.

ACTINOMYCOSIS OF THE MOUTH.

Actinomycosis of the mouth is not very common.² When it affects the *lips* it produces a tumor-like formation resembling carcinoma. The *cheek* is not often the seat of the disease. In a case of Kaposi's, the trouble began on the inner side of the cheek corresponding to the position of a carious tooth removed a short time before. In the *jaw*, the fungus commonly attacks the periosteum, producing a periostitis alveolaris, especially in the lower jaw. In the *tongue* the disease assumes a tumor-like appearance, the nodules ranging in size from that of a pea to that of a chicken's egg; they are found especially near the tip of the organ.

The cause of actinomycosis is the actinomyces fungus, which is probably conveyed to the mouth clinging to grain. There is no proof of contagion in man from animals. Carious teeth are a predisposing cause by favoring stomatitis, which in turn renders infection with the fungus easier. The cavities in decayed teeth have been suspected to be the breeding places of the actinomyces, but this is not proved.

The actinomycotic process is an infectious granuloma characterized by proliferation of connective-tissue cells and leukocytic infiltration. Interspersed among the granulation tissue are the fungi, with their striking radiate arrangement. There is a tendency to suppuration, but also to scar formation, so that in case of the mouth there are usually nodular or band-like areas of hard cicatricial tissue that are quite characteristic.

Treatment.—Excision of the diseased area, when possible; when not, the use of potassium iodide; arsenic and tuberculin, which have been tried, are of doubtful value.

LEPROSY OF THE MOUTH.

This affects the mouth and tongue only secondarily. It appears in the form of nodules, which, in the tongue, may be symmetrically situated on both sides of the raphé; by their coalescence, extensive infiltrating plaques may be formed. Ulceration is exceedingly rare and is painless; it is accompanied by salivation. The lesions are like those of leprosy elsewhere.

SCLEROMA OF THE MOUTH.

This reaches the mouth through the posterior nares, and produces infiltrated areas covered with a normal, somewhat bluish-red mucosa. After a

¹ *Loire méd.*, March 15, 1894, No. 3.

² *Centralbl. f. die Grenz. der Med. u. Chir.*, 1900, Band 3, iii, p. 561.

time very hard nodules appear that are not usually spontaneously painful, but somewhat tender on pressure. They scarcely ever suppurate, but may become superficially ulcerated.

Histology.—The infiltration consists of two layers—an upper vascular layer, and a lower rich in cells and traversed by marked fibrous bands, to which the extreme hardness is due. The similarity to a round-celled sarcoma is quite marked.

THE TONGUE.

The coating of the tongue hardly has the diagnostic value attached to it by the older clinicians. Nevertheless, it gives much information and a study of it should not be neglected; to quote Lasègue,¹ “La langue ne denonce pas la maladie, mais l'état du malade qui en est atteint.”

The coating of the tongue may depend upon conditions local in the mouth or nasopharynx; upon causes arising from the gastro-intestinal canal; or upon general conditions. A heavy coating of the tongue is compatible with good health, and the clean tongue is not always a sign that the alimentary canal is performing its functions properly. Some persons get a furred tongue on slight provocation, while others, even in severe affections, do not. A clean tongue under conditions that cause a coating in most persons is sometimes a family idiosyncrasy.

An exclusive milk diet nearly always produces a coating of the tongue, probably chiefly because of the suspension of the masticatory movements. In some persons even a single glass of milk will give rise to this condition. A coated tongue is found in mouth breathers in fevers, and in some forms of dyspepsia. In hyperchlorhydria the tongue is usually dark red, moist, and clean; it is also at times remarkably clean and “beefy” in diabetes mellitus. It is pale and heavily coated in anacidity (Rollin²).

Anomalies of the Tongue.—*Aglossia*, a total absence of the tongue, is extremely rare. There is indeed only one indubitable case, that of Jussieu (Rosenthal³).

Microglossia, smallness of the tongue, is likewise rare.

Macroglossia, large tongue, is usually applied to enlargements of the tongue not due to acute inflammation, and histologically is of three varieties: (a) Lymphatic (lymphangioma); (b) Cavernous (hemangioma); (c) Muscular or parenchymatous, the rarest, of which Helbing⁴ reports a case. A chronic enlargement, due to syphilis, might be added to the foregoing. *Macroglossia* is generally bilateral, but may be unilateral, as in the case of Little,⁵ in which it was probably due to congenital syphilis; and in that of Maas, in which it was associated with congenital hypertrophy of one-half of the body. The chief causes of *macroglossia* are cretinism and syphilis; the former producing especially the lymphangiomatous form, which may be associated with *macrocheilia*, large lip.

Doubling of the tongue, excessively long tongue, and *bifid tongue* have been described, but are unimportant.

¹ *Traité de Path. générale*, iv, p. 578.

² *Berl. klin. Woch.*, 1906, Nr. 18.

³ *Die Zunge*, 1903, p. 17.

⁴ *Jahrb. f. Kinderh.*, 1895 to 1896, xli, p. 442.

⁵ *British Journal of Dermatology*, 1906, xviii, p. 181.

Congenital deformities of the frænum are supposed to be common, and to the so-called tongue-tie the laity ascribe delayed speaking and difficulty in sucking. Cutting of the frænum is, however, very rarely necessary, although Makuen¹ has reported a case of rapid speech development in an adult following the operation for tongue-tie.

Tongue swallowing may occur if the frænum is too long. The tongue may roll back upon itself and so press against the posterior wall of the pharynx that the entrance to the larynx is occluded and suffocation results.

Tongue sucking is especially common in Mongolian idiots. It leads to marked fissuring of the tongue and to enlargement of the papillæ.

Scrotal tongue (langue scrotale) is a term much used by the French to describe a fissured tongue resembling the scrotum in its roughness. It is difficult to find an explanation for it, although sometimes there is a history of gastro-intestinal disorders. Some of the patients in early life were arthritic subjects. The condition may run in families.

Black Tongue.—*Synonyms:* Lingua nigra; nigrities lingue; hyperkeratosis of the tongue; melanotrichia of the tongue; melanoglossia; German—Haarzunge. This is a condition in which a brownish or black patch appears on the posterior portion of the tongue, in the neighborhood of the circumvallate papillæ. It is either smooth or covered with hair-like processes varying in length up to 0.5 cm. The hairs are elongated filiform papillæ that have become pigmented and cornified. Some hold that the black discoloration is due to a mould; but Blau found no mould in four cases. The causes of black tongue seem to be various. It has been found in syphilis, diabetes, neuralgia, etc. It may come and go without treatment, and this suggests that it depends upon changes in the salivary secretion. There are usually no subjective symptoms.

Varicose Veins of the Tongue.—Phlebectasiæ lingue. Dilatation of the ranine veins of the under surface of the tongue is not uncommon; but the veins on the dorsum may also become varicose, especially those running from the base forward to the circumvallate papillæ. The causes are probably local.

Hemorrhage from the Tongue.—Bleeding tongue, glossorrhagia. This is most often traumatic, but may occur in purpura and other hemorrhagic conditions, and in hysteria, of which the writer has seen a striking example. (See Plate V.)

Paralysis of the Tongue.—Glossoplegia. This may be due to central or peripheral lesions of the nervous system, and is either unilateral or bilateral. Unilateral glossoplegia may be caused by disturbance of one of the hypoglossus nerves; it is also present in the majority of cases of cerebral hemiplegia. The tongue is deflected toward the paralyzed side. The chief cause of bilateral glossoplegia is progressive muscular atrophy.

Spasm of the Tongue.—The tongue participates in the general muscular commotion in epilepsy, chorea, etc. Spasm limited to the tongue, a sort of tic, has been described; spasm of the facial and masticatory muscles may be associated with it. Pappenheim² has reported a case of glossal spasm in an alcoholic subject, which he attributes to a cortical lesion. Some cases of spasm are hysterical; in some no definite cause is discoverable.

¹ *New York Medical Journal*, 1895, 20.

² *Wien. klin. Woch.*, 1907, p. 165.

PLATE V



Bleeding Tongue in Hysteria.

Paralysis of the Soft Palate.—This, like glossoplegia, may be unilateral or bilateral. In the unilateral form the palate on the affected side hangs lower than on the normal side. In bilateral palsy the entire palate droops and is motionless during speaking and breathing; speech and swallowing are disturbed, and food regurgitates through the nose. The most important causes are neuritis following infectious diseases, especially diphtheria, scarlet fever, typhoid fever, and variola; bulbar palsy, tumors at the base of the brain in the posterior fossa; aneurism of the vertebral artery; and tabes dorsalis.

INFLAMMATION OF THE TONGUE.

Glossitis.—Acute inflammation may be due to biting of the organ during an epileptic convulsion, biting by insects, burns, or to the irritation of a rough, carious tooth. An acute glossitis is often present in smallpox, erysipelas, scarlet fever, and typhoid fever, and usually accompanies severe forms of stomatitis. Tobacco may also cause glossitis, with or without ulceration, and is responsible for the so-called smoker's patch.

Symptoms.—In traumatic glossitis the affected side of the tongue swells and at the point of injury becomes covered with a grayish-white membranous deposit. The swelling may be so great that the tongue hangs out of the mouth. Pain and discomfort are severe and mastication may be impossible. The saliva is increased in amount and is sticky. The condition usually terminates in resolution; but in about one-third of the cases, according to A. B. Bennett,¹ suppuration ensues. When unilateral in the beginning (hemiglossitis), it rarely spreads to the other side.

Treatment.—Antiseptic mouth washes are indicated. In severe cases, and or when abscesses form, a long, deep incision must be made into the substance of the organ.

Papillitis Lingualis; Linguopapillitis.—This is a painful condition of the tongue, which reveals nothing to the naked eye, but the magnifying lens shows small, ulcerating points hidden in the folds of the mucosa about the fungiform papillæ of the tip and margin of the tongue.

Treatment.—The painful points should be touched with the galvanocautery aided by a lens.

Ulcer of the Frænum.—Ulcer is most common in children suffering from whooping-cough, and is due to the forcible protrusion of the tongue and impact of the frænum against the teeth during the paroxysms of coughing.

Glossitis Papillosa Acuta; Glossitis Papillaris.—This is an inflammation limited to the circumvallate papillæ, and is characterized by slight difficulty in swallowing, burning, and sticking sensations in the throat, and an irritative cough. An acute form has been described by Michelson.² Stetter,³ under the name of glossitis papillaris, reports four cases with histological studies. Tuberculosis, as Stetter shows, may produce conditions very similar to the ones described.

Wandering Rash of the Tongue.—*Synonyms:* Geographical tongue; exfoliatio areata lingue; état lichenoïde; annulus migrans; Zungenfratt; état tigré; glossitis exfoliatricans marginata; desquamation en aires. This

¹ *Washington Med. Ann.*, 1906, v, p. 667.

² *Berl. klin. Woch.*, 1890, p. 1094.

³ *Arch. f. klin. Chir.*, 1898, lvi, p. 324.

interesting condition of the tongue is characterized by circinate, crescentic, or circular patches in which the epithelium is desquamated. The areas which are most common on the dorsum, rare on the margin of the tongue, are bordered by an elevated whitish ring, which is sometimes double-contoured. One-half of the tongue or the whole surface may be affected, the fantastic shapes of the patches giving to the organ a very remarkable appearance, well-described in the name geographical tongue. The patches may come and go suddenly; their shape and size alter rapidly; contiguous patches may fuse. The condition is most common in children, but also occurs in adults, the writer having seen it in a woman aged sixty-eight years.

Etiology.—The cause is not known. Gastro-intestinal disorders and heredity are probable factors. Syphilis, although often suspected, is not responsible for the condition.

Histological examination shows a superficial desquamation of the epithelium, the latter being replaced by a coagulated exudate containing leukocytes and degenerated epithelial cells. The cells of the deeper layers of the mucosa are in a state of cloudy swelling. Some authors have found the changes most marked in the lower strata of the mucosa.

Symptoms.—There are no subjective symptoms of moment, but the patient whose attention has been called to the condition may become hypochondriacal. Sometimes the lymph glands are enlarged, and in women the condition is apt to be worse at the menstrual periods.

Treatment.—This is not of much avail, and hence is scarcely necessary. Unna recommends a mixture of sodium hyposulphite and glycerin, of each 10 parts, and water, 180 parts.

Moeller's Glossitis; Glossodynia Exfoliativa; Chronic Superficial Glossitis.—This is a very painful affection of the tongue, characterized by bright-red lines or patches at the margin and tip. The pain, which is the principal symptom, is out of proportion to the local lesion, and is much increased by chewing and speaking. The disease chiefly affects women, especially those who are weak and decrepit.

The cause is unknown. In two of Michelson's¹ cases the patient had tape-worm. The only instance seen by the writer was in a woman with pernicious anæmia. Local treatment was of no avail; the x-ray and applications of nitrate of silver were faithfully tried, without effect. With an improvement in the blood the condition subsided, but recurred; the patient eventually died of anæmia.

Glossodynia.—Pain of the tongue, without gross lesion to account for it, is not uncommon; usually the pain is referred to the anterior third of the organ, or along the margin of the tip. It is often difficult to ascertain the cause; but in a search for it the classification of Chaveau² may prove helpful. He names the following varieties:

1. Glossodynia secondary to trigeminal neuralgia, especially of the inferior dental branch of the trigeminal.
2. Glossodynia of the insane, usually starting as a local paræsthesia.
3. Glossodynia of tabes, corresponding to crises in other organs.
4. Glossodynia of hysteria.
5. Rheumatism of the lingual muscle or rheumatic glossodynia.

¹ *Berl. klin. Woch.*, 1890, Nr. 46.

² *Arch. gén. de Méd.*, 1900, ann. 77, n. s., iii, p. 66.

6. Glossodynia attributable to local causes. These may be extrinsic or intrinsic. Among the extrinsic causes are (a) dental affections and artificial teeth (causing papillary hypertrophies and other lesser lesions); (b) granular pharyngitis and hypertrophy of the posterior pillars, and of the lingual tonsil, etc. Among intrinsic causes are mentioned (a) lingual varices; (b) chronic glossitis from tobacco, alcohol, spices, iodine, lead, gout, etc., and especially (c) papillary hypertrophy of the foliaceous region of the tongue.

Leukoplakia.—*Synonyms:* Leukoplasia; psoriasis linguæ; tylosis linguæ; ichthyosis buccalis; keratosis buccalis; smoker's patch.

This is a chronic disease of the mucous membrane of the tongue, rarely of the lips, cheek, palate, and gums, characterized by thick, elevated, indurated, whitish patches having either a fissured or more rarely a smooth appearance.

Hunter and Babington¹ allude to a condition that we should probably now call leukoplakia. Plumbe² is usually credited with being the first to describe a case under the name of ichthyosis linguæ, but in reality quotes Rayer; Kaposi, in 1866, called the condition *keratosis mucosæ oris*; Bazin, in 1868, designated it as *psoriasis buccalis*; and Schwimmer,³ in 1887, as leukoplakia; Leloir changed this to leukoplasia. Other terms met with are tyloma and hyperkeratosis.

The frequency of the disease is difficult to estimate. Schwimmer, in nine years, during which he saw 5000 cases of disease of the skin observed it twenty times; 54 cases were seen in nine years in Joseph's clinic in Vienna.

Etiology.—There is a connection between syphilis and leukoplakia, but that the former is the cause cannot be maintained, in view of the investigations of Erb⁴ and Neisser.⁵ Nevertheless, the frequency of a history of syphilis, in 65 per cent. of the cases of leukoplakia, according to Schoengarth,⁶ and in Pulvermacher's⁷ 54 cases, seem to justify the contention of Gaucher, Sergent, and Pulvermacher that leukoplakia is a parasymphilitic lesion comparable to locomotor ataxia. Smoking is also an important factor, and explains in a measure the predominance of the disease in the male sex. Of Pulvermacher's 54 cases, only 1 was a woman. The use of spices, condiments, and alcoholic beverages must be mentioned among the possible causes, likewise irritation by carious teeth and badly-fitting devices. As leukoplakia has often been found associated with diseases of the skin characterized by scaling and keratosis, it may be looked upon as an expression of a general tendency to hyperkeratosis, to which the mouth is excited by frequently repeated or long-continued irritation of various kinds. The most common dermal association is psoriasis.

As already mentioned, the disease is most common in the male sex and in adult life, although Hartzell reports a case of extensive leukoplakia associated with keratosis of the skin in a young girl aged eleven years. The disease progressed rapidly until at the age of twenty-six there was carcinoma of the tongue.

¹ Quoted by Schwimmer, *Vierteljahr. f. Dermatol. u. Syph.*, 1877, p. 523.

² *Practical Treatise on Diseases of the Skin*, 1837, p. 514.

³ *Vierteljahr. f. Dermatol. u. Syph.*, 1877 to 1878.

⁴ *Münch. med. Woch.*, 1892, Nr. 42.

⁵ Quoted by Mikulicz and Kümmel, *Die Krankheiten des Mundes*.

⁶ *Ueber Leukoplakia lingualis et buccalis; Diss.* Breslau, 1896.

⁷ *Wien. klin. Woch.*, 1906, xiii, p. 963.

Pathology.—The principal feature is a hyperkeratosis of the mucous membrane. Under the microscope, the epithelium, especially the corneous layer, is greatly thickened. The subcorneous stratum contains many cells filled with eleidin droplets and granules. Mitotic figures are abundant in the deeper layers of the rete mucosum; while the elongated papillary processes are extensively infiltrated with leukocytes.

Relation to Epithelioma.—The transformation of leukoplakia into cancer is indubitable. Carcinoma is not a complication or an accident, but a distinct evolutionary development, the causes of which reside in the histogenesis of leukoplakia. It occurs in about 20 per cent. of the cases.

Symptoms.—In the early stages leukoplakia appears in the form of deep-red erythematous patches, which after a short time assume a bluish-white color, looking as if the mucous membrane had been painted over with a strong solution of silver nitrate. Gradually the patches become thicker and larger, and, in the case of the tongue, may cover the mucosa so completely that the papillæ are no longer visible. The parts affected are in the order of frequency: tongue, lips, cheek, gums, and palate. The pharynx and the base of the tongue posterior to the circumvallate papillæ are rarely involved. The patches may be smooth, more often they are fissured. To the touch they have a tough, leathery feel. The superficial layer can at times be picked off easily without much bleeding being produced; regeneration quickly takes place.

The subjective symptoms are slight, unless the fissures are deep and raw, in which case there may be pain on chewing. The patient who is aware of his leukoplakia is apt to become hypochondriacal, fearing either cancer, or that a syphilis he had long ago, is returning under a new form.

Treatment.—This avails but little, and it is doubtful whether it is possible to prevent by any form of application the development of carcinoma. Cleanliness of the mouth, cessation of smoking, and the removal of sharp-edged teeth are important. Antiseptic treatment and strong caustics more often do harm than good. A 10 per cent. solution of tincture of iodine has been recommended, and Hartzell advises applications of salicylic acid (1 to 100 or 150). The x-ray cautiously employed has been of some value in the hands of Hartzell and Schamberg (personal communication). Bockhart¹ insists that leukoplakia cannot be cured unless the patient absolutely stops smoking. In five cases he had success with the following local treatment: the diseased areas were rubbed once a day or once in two days with balsam of Peru; in addition the mouth was rinsed from six to twelve times a day with from $\frac{1}{2}$ to 3 per cent. salt solution. He considers the saline washing more important than the application of the balsam. Antisyphilitic treatment is not only useless but often harmful. Local astringents, such as myrrh, are injurious.

Riga's Disease.—*Synonym:* *Produzione sottolinguale*. This is a benign tumor-like excrescence on the lingual frænum of infants. First described by Cardarelli in 1857, it was more fully studied by Riga in 1881. It is most common in Italy, whence nearly all the observations and studies have come. Good accounts, with bibliographies, are found in the articles of Ernst Deutsch² and E. Audard.³ Many names have been given to it

¹ *Monats. f. Prakt. Dermat.*, 1902, xxxiv, p. 164.

² *Arch. f. Kinderh.*, 1905, xl, 168.

³ *Rev. mensuelle des mal. de l'enfance*, 1902, xx, 49.

in addition to the two mentioned above—aphte cachectique; subglossite diphthéroïde; maladie de Fede. Some writers have described a malignant form leading to cachexia and death; but Deutsch thinks that the serious gastro-intestinal symptoms present in such cases are an accidental concomitant. The cause is in dispute, but in some cases it is of mechanical origin (decubital), due to irritation by the lower incisor teeth. Histologically, the features of the condition are an hypertrophy of the submucosa with papillary excrescences, and keratinization of the surface; in other words, it is a papillomatous growth.

Symptoms.—The disease appears between the ages of three and eighteen months and is equally common in both sexes. It is probably not contagious; some Italian writers, however, consider it so. It lasts from two weeks to two months; although some cases are on record that persisted for from eighteen to twenty months. It must be distinguished from the ulcer of whooping-cough, chickenpox, syphilitic ulceration, herpes, and impetigo.

Treatment.—If a cause is discoverable, it should be removed. If there is irritation by teeth, they should be extracted. In Deutsch's case, cure followed the weaning of the child. In persistent cases, caustics, silver nitrate or tincture of iodine, may be applied, or the growth may be excised.

TUMORS OF THE TONGUE.

Fibroma, usually of the hard variety, is found on the surface or in the substance of the organ. *Sarcoma* is a comparatively rare tumor, generally spindle-celled, and occurs in the substance of the tongue.

Thyroid Tumors.—A small tumor is sometimes found at the base of the tongue in the region of the foramen cæcum, which histologically is made up of tissue closely resembling that of the thyroid gland and the origin of which is to be traced to the remains of the thyroglossal duct. Glas¹ reports a case of adenomyoma occurring in the same region as the thyroid tumor, which he traced to the remains of the ductus lingualis.

Carcinoma is the most frequent tumor found in the tongue. It is commoner in the male sex (66 to 3 in the statistics of Meller), and extremely malignant. Patients not operated on usually die in less than a year after the appearance of the first symptoms.

Amyloid tumor of the tongue is a rare condition, of which an example is recorded by Gross.²

THE SALIVARY GLANDS.

The normal saliva is composed of the secretions of the salivary glands—the parotid, submaxillary, and sublingual—and of the mucous glands of the mouth. It is a watery, opalescent, slightly stringy fluid, of a feebly alkaline reaction, and has a specific gravity of from 1002 to 1008. Its principal organic constituents are mucin—a diastatic ferment—ptyalin, maltase, oxydases, and traces of proteid. Urea, according to some authorities, is also present. Among the inorganic constituents are potassium and sodium

¹ Berl. klin. Woch., 1905, 18, 746.

² Deut. Zeit. f. Chir., 1906, 84, 462.

chloride, potassium sulphate, sodium carbonate, calcium carbonate and phosphate, potassium sulphocyanide, and carbon dioxide. The total solids amount to from 5 to 10 parts per 1000. The freezing point is 0.20°C . Apart from the fact that the proteid is a glycoproteid, that is, a compound of proteid and a carbohydrate radicle, nothing definite is known of its nature. The potassium sulphocyanide content (Rhodanalkali of the Germans) varies considerably. In smokers it is said to be greater (0.2 per 1000) than in non-smokers (0.03 or 0.04 per 1000).

The secretion of the saliva is under the control of two sets of nerves: one coming from the brain, the other from the sympathetic system. In the case of the submaxillary gland, the cerebral impulses are conveyed by fibers of the facial nerve running in the chorda tympani. Studies on animals, especially on dogs, have shown decided differences in the saliva following separate stimulation of these two types of nerve fibers. The *sympathetic saliva* is scantier, thicker, and richer in solids, particularly in mucin, than the *chorda saliva*. A third variety of saliva is the so-called *paralytic saliva*, which follows poisoning with curare, or section of the gland nerves. The normal quantity of saliva varies from 1400 to 1500 grams in twenty-four hours.

The reaction of the saliva, usually alkaline, is subject to variations. It may be acid or neutral even in health; in diabetes, fevers, and dyspepsia it is often acid. In making tests, especially in hospitals, the possibility of acid mouth washes being used should not be overlooked. The relative proportion of organic as well as inorganic constituent changes in response to variations in the stimulus. Von Zebrowski has shown that the quality of the excitant, the intensity of its action, as well as the locality and manner of its application, are of importance. The secretion of saliva is stimulated more by those substances that require longer chewing. In unilateral mastication, the gland on the active side is especially stimulated; the opposite gland also secretes, but more slowly, and the saliva contains a somewhat larger amount of organic substances.

Stern and Lederer,¹ in a study of the saliva in 158 cases of diabetes mellitus, found the quantity increased in 8, diminished in 89, normal in 63. The reaction was acid in 47, alkaline in 92, neutral in 8. Glucose was found in 85. The diastatic quality was unchanged in 90 per cent. When the sugar disappeared from the urine it also disappeared from the saliva. In acute gastritis an acid secretion was found in 8 out of 20 cases, being caused in 2 by lactic acid and in 2 by acetic acid. In hyperchlorhydria an increase of secretion was found in 27 out of 182 cases; the reaction was acid in 71, alkaline in 41, neutral in 5, amphoteric in 65. The acid reaction was due to lactic acid in 12, to acetic acid in 12, to lactic and acetic acids in 9, to hydrochloric acid in 6. In pyloric stenosis the reaction was acid in 5, alkaline in 8; in hypochlorhydria the reaction was acid in 2, alkaline in 15, and amphoteric in 6. Urea, according to some authorities a normal constituent of saliva, is increased in grave forms of nephritis and in uræmia.

QUANTITATIVE DISTURBANCES OF SECRETION.

1. **Increase of Salivary Secretion** (hyperptyalism, ptyalism, salivation, sialorrhœa, sialosis) is often apparent rather than real, the saliva,

¹ *Journal of the American Medical Association*, 1904, xliii, p. 1765.

normal in amount, not being swallowed. This is the case in paralysis of the muscles of deglutition, or of those of the lips and tongue; in quinsy and other forms of tonsillitis, and in severe types of pharyngitis, when swallowing is painful.

True hyperptyalism is a condition in which more than the normal amount of saliva is secreted. Its causes are various, and may be classified as follows:

(a) *Local*.—The different forms of stomatitis; dentition; and chorea, by reason of the active masticatory movements.

(b) *Reflex*.—Hyperchlorhydria; gastric ulcer; nausea; pregnancy; trigeminal neuralgia; helminthiasis; pain from various sources. In a pregnant woman under the writer's observation the saliva collected in a bottle amounted to 300 cc. in twenty-four hours. It was acid in reaction, strongly amylolytic, and contained sugar and albumin. The patient was obliged to go around all day with a handkerchief held to her mouth to catch the drooling saliva. She was pregnant for the first time, and during the course of a few weeks lost twelve pounds in weight.

(c) *Direct nerve stimulation*, either central or peripheral; migraine; bulbar palsy; uræmia; certain poisons, as, for example, mercurial poisoning. The salivation in the last-named is only in part due to stomatitis, for it often precedes inflammatory changes in the mouth. Certain drugs, such as pilocarpine, act through direct stimulation of the nervous system. Hysterical and neurotic salivation, which in some cases depend on direct nervous influences, may also be reflex, the ordinary stimuli sufficing in an unstable nervous system to produce salivation.

Salivation in Children.—Salivation is normal in infants; it is increased by dentition, but is not dependent upon it. Salivation is present in all forms of stomatitis, and, without any local changes in the mouth, in cretinism, idiocy, and at times, as already mentioned, in helminthiasis. Jordan reported two cases of "idiopathic salivation" in children. The dribbling began during dentition and continued, without any apparent cause, after the process had stopped.

2. **Diminution of Secretion** (hypoptyalism, aptyalism, absence of secretion) occurs in fevers, especially in pneumonia, typhoid fever, and septic fevers, after the use of atropine; in fright and excitement (the rice ordeal); in loss of fluid through diarrhœa or the elimination of dropsical effusions; in cirrhosis of the liver when ascites is forming, and in atrophy of the salivary glands. The writer has also noticed an annoying dryness in arteriosclerosis, with great hypertension and polyuria, with and without thirst.

XEROSTOMIA.

Under the name of xerostomia, or dry mouth, Jonathan Hutchinson¹ described a condition of distressing dryness due to suppression of the salivary secretion. It is most common in women (32 women to 4 men, in Hall's table²), and usually resists treatment. In a case of the writer's,³

¹ *Clin. Soc. Trans.*, 1888, xxi, p. 180.

² *Quarterly Medical Journal*, 1898 to 1899, vii, p. 26.

³ *Philadelphia Polyclinic*, 1896, v, 10.

a woman aged thirty-eight years, the dryness had lasted two years, and was so great that the patient had to rise two or three times a night to wet her mouth. The urine amounted to two liters a day, had a low specific gravity, 1008, and was free from albumin and sugar. There were also darting pains in the tongue (glossodynia), and her speech was somewhat disturbed. In other cases reported, mastication and swallowing were affected. In Lartigau's case¹ a marked diminution in cutaneous activity was associated with the dry mouth; a curious association, in view of the fact that the skin and salivary glands are totally unrelated anatomically and physiologically. The tongue and mouth in xerostomia are dry, red, and glazed, and there seems to be a suppression not only of the salivary secretion, but also of that of the buccal glands. In Hall's statistics of 39 cases, 18 were over fifty years of age, 8 between forty and fifty, 5 between thirty and forty, and 3 between twenty and thirty. The tongue and mouth were affected in 36 cases, the nose also in 10, and the eyes in 7. In some cases there was a distinct history of crumbling away of the teeth at the onset of the disease. In 8 cases the parotids were enlarged. The disease began suddenly in 6; in 6 it followed mental shock, and in 4 influenza or other slight febrile affections.

Etiology.—The condition is in the majority of cases best explained on the basis of a disturbance of the nerve centres controlling the secretion of the salivary and buccal glands. In some instances it may be due to a primary affection of the salivary glands, since it is present in the so-called Mikulicz's disease. Zagari² found atrophy of the parotid and submaxillary glands (histologically determined) in a case of xerostomia in a woman aged fifty years, who had died of a gradually increasing emaciation and marasmus. Zagari attributed the marasmic state to lack of an internal salivary secretion.

Treatment.—The condition has generally proved rebellious to treatment. The galvanic current is worth a trial, and pilocarpine in small doses may also be employed.

INFLAMMATION OF THE SALIVARY GLANDS.

Acute Inflammation.—Acute inflammation of the parotid gland—parotitis—appears in two forms: the epidemic parotitis or mumps, and the so-called secondary parotitis, which is found associated with typhoid and septic fevers, pneumonia, dysentery, pyelitis, smallpox, measles, scarlet fever, and is also seen at times after abdominal operations, especially oöphorectomy. As far as the writer's observation goes, it is most common in diseases situated below the diaphragm. In obscure febrile cases the appearance of a parotitis should lead the physician to consider carefully the possibility of typhoid fever, abscess in the pelvis of the kidney, or a perinephritic abscess. The condition has also been called splanchnic parotitis.

A parotitis occurring in the course of hemiplegia has been described by Gilbert and Villaret.³ In both cases observed by them the parotid on the paralyzed side was affected.

¹ *Medical News*, 1898, lxxiii, p. 548.

² *Il Policlinico*, 1907, xiv, Medical Section.

³ *Sem. méd.*, 1906, p. 104.

Iodic Parotitis.—The writer has seen an acute enlargement of the parotid gland following the administration of potassium iodide; it may also occur as a complication of lead poisoning.

Chronic Inflammation.—Chronic inflammation of the salivary glands usually depends on retention of salivary secretion, and is most common in the parotid. The gland is enlarged and hard, and may resemble a malignant tumor, as in three cases reported by Steinhaus.¹ The disease is attributable to an infection propagated from the buccal cavity, and is often associated with the presence of stones in the ducts or the glands themselves. The size of the gland varies according to the degree of obstruction and retention.

Sialodochitis Fibrinosa.—Under this name Emden² reports the following case: A woman, aged fifty-four years, suffered at first in long intervals, later, regularly every eight days, from bilateral swelling of the submaxillary and sublingual glands. The attack began with burning pain in the tip of the tongue, was unaccompanied by fever, and produced a firm, dense infiltration of the glands. The whole process subsided after three days, with expulsion from the salivary caruncles of fibrinous plugs, and with profuse salivation. The plugs measured several centimeters in length and branched dichotomously. Emden compares the condition to fibrinous bronchitis, and, while not denying the possibility of a secretory neurosis, attributes it to inflammatory changes in the salivary ducts. Analogous cases have been reported by Naegeli-Akerblom,³ and by Viaud,⁴ who coined the term "whartonitis."

TUBERCULOSIS OF THE SALIVARY GLANDS.

The salivary glands are not often attacked by tuberculosis, a comparative immunity they share with the pancreas. Even experimentally, infection of the glands is not easily accomplished unless their nutrition is first disturbed by trauma. In man tubercle bacilli may reach the glands through the blood, the lymphatics, the excretory duct, or by direct extension from neighboring parts. It is still a question, however, whether in the so-called primary form of tuberculosis the disease is not located in a lymph gland occasionally found embedded in the parotid, from which the disease extends to the parenchyma proper. Mintz⁵ collects 8 cases of so-called primary tuberculosis affecting the parotid; and O'Zoux⁶ found 4 of the submaxillary. Legueu and Marien, finding in their cases chiefly parenchymatous lesions, concluded that the infection had been propagated along the duct. Bockhorn,⁷ Mintz,⁸ and Lecène⁹ believe in lymphatic transmission, the principal lesions in their cases having been found in the intra-acinous connective tissue. The infection, whether it travels by the one route or by the other, usually occurs through

¹ *Zeit. f. Heil.*, 1905, xxvi, *Abth. f. path. Anat.*, p. 194, 201.

² *Sitzungsbericht des aerztl. Vereins in Hamburg*, 1897.

³ *Monats. f. Ohrenheilk.*, 1895, Bd. xxix, S. 80.

⁴ *Thèse de Paris*, 1894.

⁵ *Deut. Zeit. f. Chir.*, 1901, lxi, p. 290.

⁶ *Arch. clin. de Bordeaux*, 1897, vi, p. 28.

⁷ *Arch. f. klin. Chir.*, 1898, lvi, 189.

⁸ *Deut. Zeit. f. Chir.*, 1901, lxi, 290.

⁹ *Rev. de Chir.*, 1901, xxiii, 524.

carious teeth or ulcerated gums about such teeth. Tuberculosis of the salivary glands is of slow development, and is generally mistaken for the so-called mixed tumor. The histological changes are those found in tuberculosis of other organs.

SYPHILIS OF THE SALIVARY GLANDS.

Syphilis of the salivary glands is rare, and has hitherto been found principally in the sublingual gland. It is usually of the tertiary form, and in the differential diagnosis, which may be difficult, the history and the therapeutic test give the most assistance.

SYMMETRICAL ENLARGEMENT OF THE LACRIMAL AND SALIVARY GLANDS—ACHROÖCYTOSIS, MIKULICZ'S DISEASE.

The first complete description of this was given by von Mikulicz.¹ It is a chronic inflammation affecting the salivary and lacrimal glands; but there are cases, evidently of the same nature, in which the salivary glands alone are affected. Blood changes are rare; in some cases, however, the blood presents the picture of leukæmia or pseudoleukæmia. The lymphatic glands may be enlarged and at times there are analogous infiltrations of the skin. The enlargement of the salivary glands is due chiefly to a diffuse lymphoid hyperplasia, and not to hypertrophy of the parenchyma proper. Von Brunn is of the opinion that the disease is due to some infectious agent carried to the glands in the blood stream. Napp² describes a case of Mikulicz's disease in a woman aged twenty-seven years, in whom in addition to the enlargement of the lacrimal, submaxillary, and parotid glands there were submucous nodules on the lips and cheeks and in the conjunctiva. In the conjunctival nodules, which were the only ones examined, tubercle bacilli were found. On the strength of this case, Napp accepts the view of Meller that Mikulicz's disease is not a disease *sui generis*, but a symptom complex that may be produced by a variety of causes, such as leukæmia, pseudoleukæmia, atypical lymphomatosis (sarcoma), and tuberculosis. For the treatment of the condition, Pfeiffer recommends the x-ray.

Atrophy.—In addition to the shrinkage of the glands accompanying advancing age (senile atrophy), there is an atrophy of obscure nature that may or may not affect all of the glands simultaneously. In a case of such atrophy reported by Dubreuil-Chambardel,³ the condition revealed itself by an almost absolute smoothness of the floor of the mouth, and by hollows in the region of the parotid glands. The patient, an otherwise healthy man, aged sixty-one years, complained of dryness of the mouth. The urine was increased in amount to 2000 cc. in a day, but was free from albumin and sugar. Such an atrophy may be the underlying cause in some cases of xerostomia.

¹ *Münch. med. Woch.*, 1888, p. 759; *Beitr. z. Chir., Festschrift f. Billroth*, Stuttgart, 1892, p. 610.

² *Zeitsch. f. Augenheilk.*, 1907, 17, p. 513.

³ *Province méd.*, vol. xxi, 6.

SALIVARY CONCREMENTS.

Tartar or Salivary Calculus.—Tartar is a laminated deposit upon the teeth, varying in color and density. The common sites are the lingual surfaces of the lower incisors and canines, and the buccal surfaces of the upper molars. Two varieties are described, the soft and the hard; the former being rapidly deposited, buff colored and abundant; while the latter is slowly deposited, dark, and firmly adherent to the tooth surface. Dental writers speak of serumal or sanguinary and of salivary calculus. The first is derived from the blood, is deposited under the gums, about the roots of the teeth, and contains urates. The second is made up of the precipitated salts of the saliva, and is found on the fangs of the teeth. The deposit of serumal tartar is in some way connected with gouty conditions of the system, while that of the salivary calculus is favored by the rapid escape of CO_2 , which normally holds the salts of the saliva in solution. Calcium carbonate and calcium phosphate make up the bulk of salivary calculi. Traces of iron phosphate and silica are sometimes present. Sanguinary tartar contains uric acid and urates. The deposit of tartar leads to more or less absorption of the alveolar process and to chronic gingivitis.

Treatment.—The treatment consists in the removal of the tartar, which can only be done by a properly trained dentist. Careful brushing of the teeth is the only means of retarding the accumulation of salivary calculus. Prevention of the serumal form, if it be possible, can be brought about only by attention to the general health; by regulation of the diet, by exercise, and by such measures as are generally instituted to combat the gouty diathesis.

Stones Proper; Salivary Calculi.—Stones may form in the ducts, rarely in the glands themselves, the most frequent site being Wharton's duct, at a point from 10 to 20 mm. from the salivary caruncle. They seldom exceed the size of a pea, and are composed chiefly of calcium phosphate or calcium carbonate, which constitutes from 62 to 95 per cent. of the stone. They are usually single, but in the case of Brin there were four in one duct. Duct stones are cylindrical and smooth; gland stones usually round or irregular.

Etiology.—Stones formed in the ducts may have a foreign body, such as grains, seeds, bits of food or tartar as a nucleus, but it is probable that, as in the case of gallstones, the impetus to stone formation is given by bacterial infection. Inflammatory changes in the glands, by bringing about a change in the consistency of the saliva, may lead to stone formation. Thus, in a case in the care of Dr. Walter Roberts, at the Polyclinic Hospital, Philadelphia, stone formation followed a parotitis complicating typhoid fever. The patient was a woman aged thirty-nine years, who had had typhoid fever eight years previously, and at that time there was a swelling of the right parotid gland, which was but slightly painful and subsided rapidly. After an interval of five years, the gland again became inflamed, hard, and indurated. There was a sense of constriction in the right cheek, and pain while eating. During her meals she would frequently stop and massage the duct. After a supposed stricture at the orifice had been cut, the patient was able to feel a small lump in the cheek. This was worked downward until it could be felt just above the orifice of the duct. At times the duct would discharge pus and mucus, and the gland would swell and become very painful. The

stone, which was hard, dense, and irregular in outline, was removed under anæsthesia by an incision from the buccal side. Recovery followed.

In several reported cases of stone there was a history of long-continued irritation of the mouth by a pipe.

Symptoms.—Stones located in the glands do not, as a rule, give rise to marked symptoms, although the gland may be enlarged, firm, and tender. The characteristic feature of stone in the duct is an intermittent pain along the floor of the mouth, the so-called salivary colic, which usually comes on during eating and persists for from one to several hours. It is associated with a similarly intermittent tumefaction of the gland and a distention of the proximal part of the obstructed duct. At times there is a purulent discharge (pyorrhœa salivaris). The stone can generally be detected by sounding, by digital palpation, or by means of the *x*-ray.

Salivary calculi situated on the floor of the mouth may simulate ranula, from which they may be distinguished by noting that in calculus the flow of the saliva is stopped and that a probe cannot be passed along the duct, while careful digital exploration or the *x*-ray may reveal the concretion. Malignant disease, actinomycosis, and tuberculosis must also be borne in mind in the differential diagnosis, but it is unnecessary to go into detail, as the mere mention of these conditions will suggest the points upon which a differential diagnosis should be based.

Treatment.—The treatment consists in the removal of the stone by surgical means.

TUMORS OF THE SALIVARY GLANDS.

The most interesting as well as the most important new-growth is the so-called *mixed tumor*, which has puzzled pathologists for many years. Clinically, it is a slowly growing, movable, smooth, or nodular tumor, usually painless, very disfiguring, and often attains an enormous size. It is more common in the parotid than in any other of the salivary glands, the distribution, according to Boehme, being as follows: 74.1 per cent. of all cases affect the parotid, 7.7 per cent. the submaxillary, and 1.1 per cent. the sublingual. The skin is rarely adherent. The tumors have a capsule composed of fibrous connective tissue; sometimes nodules of unchanged or inflamed salivary gland tissue are included within the capsule. From the capsule septa extend inward and subdivide the tumor into lobules. The color varies from reddish to yellowish gray. According to the amount of mucoïd or hyaline degeneration, the surface is opaque, glistening, or gelatinous; at times cystic spaces are present. If the tumor is malignant, it bursts through its capsule and infiltrates the neighboring tissues; it may cause metastasis in the regional lymph glands, but transference to distant organs is rare. The soft tumors are more malignant than the hard ones, though local recurrence is more common in the cartilaginous forms.

Microscopic studies reveal a very complicated structure, suggesting in some cases carcinoma, and in others sarcoma. Mucoïd and fatty tissue and cartilage may still further obscure the picture. In a goodly proportion of cases, 14 out of 26 studied by Ehrich, squamous epithelium is present and may show characteristic horny changes. The cells which constitute the tumor parenchyma proper are arranged in larger or smaller masses resembling gland acini, or in tubules, the lumen of which is lined with cubical or

cylindrical cells. It is the origin of these cells that has given rise to most dispute. Some authorities, especially Volkmann¹ and Kelly,² hold that the cells spring from the endothelium of the lymph spaces or perivascular lymph sheaths, and that the tumors must be classed with the endotheliomas and peritheliomas. Küttner's³ extensive studies of 97 tumors of the submaxillary gland collected from the literature lead him to the view propounded by Volkmann. He finds 64 to be endotheliomas, 6 sarcomas, 3 adenomas (?), 5 carcinomas, and 19 of doubtful nature. The endotheliomas may be benign in their clinical manifestations, or may resemble sarcoma and carcinoma in the malignancy of their course. Those not having cartilage show a greater tendency to malignancy than the enchondromas.

There are many writers, however, who contend that not only morphologically, but also histogenetically, the tumor cells are epithelial. This is the view of Landsteiner,⁴ Ehrich, Lotheissen,⁵ and others. But Kelly maintains that no connection can be traced between the apparently epithelial columns of cells of the tumors and the salivary gland proper. Given certain conditions of growth, endothelium can easily assume the shapes of epithelial cells. Those tumors in which masses of squamous epithelial tissue are found may, as many have shown, take their origin from remains of the branchial arches. Guleke⁶ believes that some of the parotid tumors are teratomas and arise from germinal displacements of the original salivary gland "Anlage;" this latter contributing the cellular elements of the tumor, while the myxomatous, cartilaginous, and osteoid tissue is derived from the remains of the first branchial arch. Wood,⁷ who has published an excellent study of the tumors of the salivary glands, is also inclined to the view that they arise from embryonic displacements of epiblastic tissue during the process of formation of the parotid and submaxillary glands and the branchial arches.

So far as it is possible to decide at the present time, those who contend for the epithelial origin of the so-called mixed tumors of the salivary glands have the best of the argument. Muroid and hyaline degeneration, which are very common in tumors of the salivary glands, produce complicated pictures that have led to additions to our terminology; thus, there is the cylindroma, the myxomatous endothelioma, etc., which need no special description.

Treatment.—The treatment is surgical.

Other tumors of the salivary glands are sarcoma, cases of which are described by Kelly, Lotheissen, and Schridde,⁸ lipoma and chondroma. Von Mangoldt describes a cavernoma—a congenital telangiectatic growth, which was found associated with angioma of the overlying skin. Bidone⁹ also reports a case of this rare tumor, and reviews the literature fully.

An echinococcus cyst is described by André.¹⁰

¹ *Deut. Zeit. f. Chir.*, 1895, xli, 1 to 180.

² *Philadelphia Medical Journal*, February, 1899.

³ *Beitr. z. klin. Chir.*, 1896, xvi, 181 to 256.

⁴ *Zeit. f. Heilk.*, 1901, Fn. ii, *Abth. f. Path. Anat.*, pp. 1 to 18.

⁵ *Beitr. z. klin. Chir.*, 1897, xix, 481 to 492.

⁶ *Arch. f. klin. Chir.*, 1906, lxxxi, 275.

⁷ *Annals of Surgery*, 1904, xxxix, pp. 57, 207.

⁸ *Beitr. z. path. Anat. u. z. Allg. Path.*, 1903, xxxiv, pp. 136 to 142.

⁹ *Arch. di Ortopedia*, 1897, xiv, p. 398; 1898, xv, p. 16.

¹⁰ *Bull. de la Soc. Anat. de Paris*, 1898, lxxiii, pp. 264, 267.

LUDWIG'S ANGINA.

This is an inflammation—a cellulitis—of the tissues of the floor of the mouth in the submaxillary region. The primary focus is, usually, according to the studies of Thomas,¹ some insignificant lesion in the mouth, as a carious tooth, tonsillitis, an ulcer, etc., the infection travelling by way of the lymphatic vessels to the submaxillary lymph glands. In 18 cases studied bacteriologically the streptococcus was found alone in 6 cases, the streptococcus associated with other organisms in 8, the staphylococcus alone in 2, the pneumococcus alone in 1, and an undetermined bacillus in 1. The male sex is far more often affected than the female.

Symptoms.—The disease usually begins acutely, with slight fever, and at times difficulty in swallowing. Coincidentally, an indurated swelling appears in the region of the submaxillary gland, on one or both sides, which soon extends along the cellular tissue toward the chin, toward the parotid, and down toward the larynx or the sternum. The sublingual tissues form a hard congested swelling arranged like a cushion just inside the inferior maxillary bone, and force the tongue upward and toward the opposite side. The skin over the swelling, during the first four or six days, is movable, then it becomes reddened, œdematous, and adherent; at times there is crepitation. Soon an opening forms in the floor of the mouth through which a thin grayish or reddish-brown fluid is discharged; at the same time the constitutional symptoms become more severe, the fever higher, sleep is disturbed, profuse sweats, delirium, and dyspnoea appear, and the characteristic typhoidal state, due to septic infection, is developed.

Death, in the majority of cases, occurs from invasion of the larynx, the lower respiratory tract, and in some cases of the lungs. The mortality is high; of the 106 cases collected by Thomas, 43 died.

Treatment.—This is surgical; the best incision being that of Delorme, over the submaxillary gland and parallel with the lower jaw.

¹ *Univ. of Med. Bull.*, March, 1908.

CHAPTER III.

DISEASES OF THE ŒSOPHAGUS.

By JOHN McCRAE, M.B. (TOR.), M.R.C.P. (LOND.).

Descriptive.—The œsophagus, the viscus which connects mouth and stomach, is understood as beginning at the upper border of the cricoid cartilage and ending at the cardiac orifice; it measures, as an average of a large number of adult cases, 24.4 cm. (about nine and three-quarter inches); at birth it measures 8 cm. (three and one-fifth inches); it begins at the level of the sixth cervical cartilage, where it lies immediately in front of the vertebræ, then it comes slightly forward, and at the seventh dorsal vertebra it bends slightly to the right, then to the left to reach the opening through which it passes the diaphragm. It enters the stomach at a point corresponding to the origin of the twelfth left rib from the twelfth dorsal vertebra, and the cardia lies about 40 cm. from the upper incisor teeth. The œsophagus is divided, for descriptive and other such purposes, into a cervical part 5 cm. long, a thoracic 18 cm. long, and an abdominal 2 cm. long. If it is desired to know the distance from the incisor teeth to the cardia, Rosenheim gives the following rule: Take a point 2 cm. below the most prominent part of the occipital protuberance, measure from it to the origin of the twelfth rib, and add 7 cm. (from uvula to upper teeth) for the total figure. It is probable that for the most part the walls of the tube lie in contact with one another, but it is yet undetermined whether or not the cardia lies open when the organ is at rest.

The œsophagus is lined by stratified pavement epithelium. In the upper part the muscularis is composed mostly of striated, and in the lower half of unstriated muscle fibers, although the demarcation is not distinct. Mucous glands in rather scanty numbers in half of all cases lie in the submucosa and open by ducts which pierce the muscularis mucosæ; glands of a different kind are found between the level of the cricoid cartilage and the fifth ring of the trachea, lying superficial to the muscularis; these glands contain not only mucous cells, but also cells similar to the parietal cells of the glands of the stomach; and at times typical stomach epithelium is found aggregated in areas of considerable size, which on superficial examination resemble shallow ulcerations, of which the true nature becomes apparent only on microscopic inspection. These are found in 3 to 6 per cent. of all cases (Glinski).¹ In the lower part of the œsophagus are the cardiac glands, which are similar to the cardiac glands of the stomach.

The arterial supply consists of an anastomotic network, which is supplied from above downward by the inferior thyroid, the posterior bronchial arteries, and the œsophageal branches of the aorta. The lower end is

¹ *Bull. de l'Acad. des Sc. de Cracovie*, November 9, 1903. See also *Schwalbe, Virchow's Archiv*, 1905, Bd. 179.

supplied by the left coronary artery of the stomach, and anastomoses occur from the inferior phrenic vessels. The venous supply consists of three main channels, of which the first leads to the inferior vena cava by the diaphragmatic veins; the second leads to the superior vena cava by way of numerous branches, the inferior thyroid, the pericardial, the posterior mediastinal, the intercostal, and the diaphragmatic veins. These last lead to the azygos or to the vena cava itself. The third channel, most important in its relation to varices of the œsophageal veins, leads by way of the left coronary vein of the stomach to the portal system. The venules leading to all three channels are closely interlaced throughout the œsophageal walls.

The lymphatics of the œsophagus lead from the upper part to the deep cervical nodes lying in the vicinity of the bifurcation of the carotid artery, and from the inferior part to the nodes of the posterior mediastinum, as far as the diaphragm.

The nerves supplying the œsophagus are, above, the recurrent branches of the vagi, and below, a plexus formed of branches direct from the trunks of the vagi, into which enter branches of the sympathetic.

The question whether or not absorption occurs from the œsophagus is not yet authoritatively settled. Meltzer declared that absorption from the healthy œsophagus is better than from the stomach; Pfaff, on the contrary, thinks that it does not occur. Probably the truth of the matter lies in this, that the œsophagus can absorb, but rarely obtains opportunity therefor.

Methods of Examination.—Palpation.—This may give information of a tumor high up in the œsophagus, and the glands connected therewith may sometimes be felt or a diverticulum full of food material may be felt, and sometimes emptied by pressure; but the application of these methods is necessarily very limited.

Percussion.—Percussion will prove useful very rarely; it may assist in the determination of a large diverticulum or tumor.

Auscultation.—Auscultation gives little satisfactory information; over the whole extent of the œsophagus, when fluid is swallowed, a gurgling sound may be heard, which may cease at the level where an obstruction exists; the sounds may be heard over the neck, down the left side of the spine, and finally at the angle made by the left costal margin with the xiphoid cartilage. Very commonly at this point, six or seven seconds after swallowing, a sound is heard which is interpreted by some as fluid passing through an almost closed or half-closed cardiac orifice, by others as a sound produced in the stomach. In some people, immediately after swallowing, a sound is heard which is taken to indicate the passage of fluid through the open cardia. So experienced a clinician as Ewald is extremely skeptical of the value of the sounds produced in the œsophagus.

Stomach Tube, Sounds, Bougies, etc.—These are very useful as instruments of examination, as well as in the treatment of œsophageal disease. The best for a preliminary examination is the soft-rubber tube, and if, subsequently, sounds are necessary, the graduated, gum-elastic English sounds, which when heated become flexible and can be bent to the desired shape. Briefly stated, the rules for all such examinations are these: Ostentatious cleanliness beforehand is necessary, so that the patient's nausea or disgust through mental channels shall not be excited; let the patient sit with the head thrown slightly forward and the chin raised; if necessary, paint the pharynx with 10 per cent. cocaine solution, and do not put the fingers in the patient's

mouth; glycerin as a lubricant may generally be dispensed with, the tube being best taken last from cold, clean water; if sounds are used, from warm water; direct the patient to take full, deep breaths, and enforce this upon him at any time he becomes excited. Contra-indications to the use of the tube are as follows: *Absolute*: Aneurism, recent hemorrhage, recent corrosion, or ulceration. *Relative*: Advanced arteriosclerosis or cardiac disease, acute œsophagitis, or cirrhosis of the liver when varices may be suspected.

Dyspnoea and cough upon introduction of the tube may suggest that the instrument has entered the larynx; if the patient can phonate aa-a or ee-e, it has not done so.

Œsophagoscope.—The findings from this, to the practised eye, are of the utmost value; the discomfort it causes the patient and the difficulty of its use by the physician have not yet allowed the widespread employment of the œsophagoscope; if possible, its use should precede any important operation on the œsophagus.

CONGENITAL MALFORMATIONS.

Malformations of the gullet are not common, and are clinically of so little importance as to deserve the merest mention; they consist, for the most part, of a series of variations dependent on the fact that embryologically the œsophagus and pharynx have a common origin, and that the continuity of the œsophagus may be interrupted by a stenosis or obliteration. Thus the tube may be open above and below, and its central part may be represented by a mere cord. Either the upper or the lower segment may open into the air passages. It sometimes happens that the œsophagus, although patent, opens into the trachea, and aspiration pneumonia is thus produced before any suspicion can arise of abnormality. Two gullets have been known to exist side by side. The anomalies that are congenital and consistent with life are referred to in different parts of this article.

INFLAMMATION OF THE ŒSOPHAGUS.

Acute Œsophagitis.—**Etiology.**—Acute inflammation of the œsophagus is a condition infrequently found at autopsy, but is probably much more frequent in life. It occurs by extension of disease from the pharynx or stomach, or from other nearby organs, is excited by mechanical irritation, such as a foreign body or a rough sound, by hot liquids, corrosives, or other chemical irritants, and occurs as an accompaniment of inflammation of mediastinal tissue or other parts near but external to the œsophagus, and in various acute diseases, such as diphtheria, scarlatina, variola, measles, pyæmia, and typhoid fever.

It is a remarkable circumstance that the œsophagus has so great an immunity to the spread of infections from the pharynx; it must have impressed anyone who has had the opportunity of seeing autopsies upon patients who have died of scarlet fever or measles, that when the œsophagus is laid open its pale healthy surface contrasts vividly with the reddened hyperæmic pharyngeal structures, and that the infection stops short at a well-defined line which marks the level at which the walls of the gullet appose. Cases

in which the inflammation spreads to the œsophagus are observed at times, but are the exception. The food and saliva must constantly wash down infective material from the mouth, but this remains only momentarily; there must, however, be another explanation, which is perhaps that the œsophagus is a strongly resistant part. Even when corrosives are swallowed it often escapes with less injury than the pharynx or the stomach, and it is certain that the constant passing of rough particles of food seems rarely to tear the œsophageal membrane, which is habituated to the insult of temporary irritation.

Special Pathology.—Many different forms and degrees of severity are recognized, as catarrhal, follicular, ulcerative, phlegmonous, exfoliative, or necrotic, the relation of which to one another is close. In the ordinary catarrhal form the wall is reddened, and there is excess of secretion with desquamation as a sequence. This is the usual result of fluid too hot or mildly irritative, not to say corrosive, or as adjunct in the vicinity of foreign body or new-growth, although here it is of little clinical significance. Such an œsophagitis is the result, too, of a passive hyperæmia from cardiac disease, although the long continuance of such a cause generally operates to place it in the class of chronic disturbance. This simple form may be associated with the exanthemata, or the membranous type may be observed where an actual fibrinous membrane, not to be confounded with the desquamating dead layers, is present. Very rarely, the membrane is truly diphtheritic, which obviously may lead to ulceration. The pocks of variola have been frequently found in the œsophagus, Wagner finding them in about 13 per cent. of a large series. Various other forms of disease are occasionally noted; in the condition termed follicular œsophagitis the mucous glands of the upper part of the tube can be distinguished as lumps, sometimes of the size of 6 or 7 mm. diameter, which exude abundant mucus through the dilated duct on pressure; microscopically, such glands are found to have undergone a mucoid degeneration, to be surrounded by a zone of inflammatory infiltration, and each area may finally form an ulcer.

Allied to this condition is the formation of retention cysts, when the thickened epithelium occludes the duct; these cysts, which may attain a diameter of 1.5 cm., have been known to become infected, and the result is the formation of abscesses, circumscribed but tending to extend their boundaries. Should this occur, we have an example of purulent or phlegmonous œsophagitis, although the etiology of the latter is generally other than this. It most frequently happens in the upper part of the tube subsequent to the lodgement of a foreign body, the action of a corrosive, or by extension of an extraneous inflammation. Several cases are recorded in which, some days after drinking lyc, mucosa and submucosa have been shed as a membrane. Dorr, indeed, found one such piece 30 cm. long, this doubtless including parts of the pharyngeal mucosa; some, with Bornikel, consider this as an undermining phlegmonous œsophagitis, which quickly loosens the entire wall; others think it a necrotic process of less intensity. The two possibilities are so nearly related that it is not of importance to which we adhere. When abscesses form they are apt to undermine the mucosa and lead to the establishment of sinuses, or even of fistulae into the respiratory tract. The possibility of the causation of peri-œsophageal abscesses in this way is also to be remembered. But the infection may also be in the other direction, and peri-œsophageal abscess may undermine from the outside, or

the disease in the œsophagus may be the result of extension from a perichondritis, a vertebral abscess, suppurative lymph nodes, or even from empyema. In such cases the mucosa may be finally perforated. Such a condition is not necessarily fatal; here belong those wonderful cases in which large pieces of the lining, sometimes the entire lining of the tube, have been cast off in one mass, although the cases were not instances of corrosive poisoning. Where a long-continued inflammation has existed, strong retching or vomiting has separated the layers, and cure is reported after a cast 13 cm. long had been so expelled (Stern). Birch-Hirschfeld reported long ago a case in which a 20 cm. cast was expelled three days after the onset of the first œsophageal symptoms; this woman promptly recovered. A remarkable series of 4 cases is reported by Zenker and von Ziemssen; in these a layer of pus had separated the layers of the œsophagus and finally eroded the mucosa in numerous places. Healing occurred with a series of chambers left in the wall communicating with the œsophageal cavity. The statement made by these authors is almost incredible that no disastrous results occurred from the lodgement of foreign bodies or the decomposition of ingesta in these spaces. In the necrotic form ulcerations occur alternating with fibrin-covered areas; on these latter an actual membrane often forms, and the ulcerations may give rise to hemorrhages; this combination has been reported as following many of the acute infectious diseases and in cases of sepsis. In the Royal Victoria Hospital autopsy series a girl, aged nineteen years, dead of septicæmia, showed in the œsophagus multiple irregular ulcerations, the result of thromboses of small vessels. One of these perforated into the left pleura, and caused a hemorrhagic pleurisy.

Corrosive Œsophagitis.—Œsophagitis from corrosives may be referred to, although it is obvious that the pathological condition will fall under one or other heading already dealt with; a single case of corrosion may combine several different forms at the same time in different parts of the tube. Where the corrosion is comparatively mild, the epithelium is whitened and converted into a rough membrane peeling off in patches, but leaving an apparently intact membrane below it. The epithelium may be colored according to the corrosive used. Thus, sulphuric acid generally blackens, carbolic whitens, and nitric acid gives a yellow color to the surface. When the corrosive is a very severe one, the mucosa is converted into a dirty gray slough, peeling off irregularly from a reddened, hemorrhagic surface, which is greatly inflamed; the muscular coat is in these cases extremely flabby and the tube sags. A little later these tissues become black and gangrenous looking. Should death not occur soon, suppuration and ulceration in all directions may happen, and at the best, healing will be attended with the highest degree of stricture.

Infective Œsophagitis.—In the specific infections, besides diphtheria and variola mentioned above, the ulcers of typhoid fever are occasionally found in the œsophagus, the latter happening once in 83 autopsies upon cases of typhoid fever in our series. Thrush is found as a result of extension from the mouth, but in most instances is of little importance. Those acute inflammations which arise as a secondary affection of the walls of diverticula, etc., differ in no way from the primary forms.

Symptoms.—When corrosives have been swallowed, the history and inspection of the pharynx generally make the case clear. There is usually severe pain along the course of the gullet, dysphagia, and expectoration of

mucus, often bloody. Thirst is extreme. In the severest cases there may be no pain. With regard to the other forms of œsophagitis, with lesions of so variable intensity, it is not easy to set down a train of symptoms that will be accurate; it is of the utmost importance to know if damage has been done to the gullet by food material or other foreign body. Apart from the history, the most dependable symptom is inability to swallow or pain in swallowing; pain may exist when the head is moved violently, especially backward, and when the œsophagus can be reached from the neck, pressure may elicit tenderness. There is often thirst, and sometimes regurgitation of swallowed material which may be coated with mucus. Hard, dry food, or very hot or very cold fluids, will give more pain than less irritating material. If a sound should be used (it is usually contra-indicated), great pain is generally caused, which is apt to remain for a considerable time after its withdrawal.

Sequelæ.—Slight degrees of inflammation are almost certain to disappear completely, and severe degrees, with ulceration, are equally certain to be followed by stenosis. Any inflammation may progress and lead to the ulcerative or phlegmonous form, which in turn may give rise, with or without perforation, to peri-œsophageal abscess. If the inflammation leads to perforation, especially low down in the œsophagus, the respiratory tract, trachea, bronchus, or pleura, is likely to be invaded by the escaping material.

Diagnosis.—œsophagitis, especially occurring in the course of some severe disease, will generally be overlooked. Excessive pain from the use of tube, sound, or œsophagoscope, should suggest the immediate discontinuance of its use and the possible existence of œsophagitis.

Treatment.—In the severe forms, such as occur in the swallowing of corrosives, oil may be given, but after this the gullet is to be left absolutely alone, and all food and drink should be given by the rectum; should this not be possible, gastrostomy may be required. If the secretion is very great, and pain and nervousness cause the patient to make constant swallowing efforts, morphine had better be employed; ice-bags may be applied to the stomach, neck, or back; hot applications also are serviceable.

Thrush requires mild antiseptic drinks. For the slighter forms of œsophagitis little treatment is necessary if it be remembered that the blandest and most mucilaginous forms of food are the best, and that all the œsophagus needs is a little time, and complete freedom from irritation; demulcent drinks are of great use; ice may be sucked and cold drinks in small quantities are grateful, and presumably do no harm; occasional draughts of bismuth emulsions are useful. The use of tube, sounds, or bougies is contra-indicated in the acute stage. The greatest degree possible of physiological rest is the solution of the problem.

Chronic œsophagitis.—It is largely an arbitrary matter as to what we designate acute or chronic œsophagitis. A recurring acute inflammation becomes chronic; a long-continued, slight inflammation, which is never severe enough to cause marked symptoms, is generally, when discovered, called chronic. We have previously suggested that the alterations we call degenerations are not essentially different from inflammations; at least, they may be admitted to end at the same goal. Therefore, reference will be made here to certain chronic inflammations, and to several conditions, such as rupture, dilatation, and diverticulum, which have some relationship to the degenerations of the œsophageal wall, whether these are actually caused by inflammation or not.

Chronic œsophagitis is found chiefly in drunkards, occasionally in heavy smokers, and as a result of extension of pharyngitis or gastritis. Occasionally it may follow a repeated chronic congestion brought about by disease of the heart or lungs. The condition is often found at autopsy, and is generally overshadowed during life by the symptoms of more apparent lesions. It is thus of itself an unimportant subject.

Special Pathology.—The form of chronic inflammation most frequently found at autopsy is that which shows a series of grayish streaks corresponding to the crests of the longitudinal folds into which the tube falls when at rest. Where the sides of the rugæ lie in apposition this membrane is not apparent, and the mucosa is generally dusky red or bluish, the attrition serving to rub off the membrane as soon as formed. The membrane appears to consist of degenerated superficial layers with little or no fibrin. This picture differs little from that of acute catarrhal œsophagitis, except that the lividity of the wall is more extreme. This low grade of disease may exist for a long time without giving rise to any symptoms, but long persistence leads to mucosal thickening and sometimes to a granular appearance of the membrane. Next to this, and closely allied to it, is the so-called leukoplakia, in which there are white, yellow, or gray plaques of irregular size and distribution, overlying and alternating with thickened and reddened mucosa and submucosa; these areas are slightly harder and more raised than the rest of the mucosa, and are underlaid by inflammatory infiltration. This is precisely the same condition that is well known in the mouth, and it is thought to have some etiological force in the causation of carcinoma, although the figures cannot be said, as yet, to be conclusive. The strongest causative factor in the production of leukoplakia of the œsophagus is alcoholism. The late Wyatt Johnston insisted strongly upon this, and Professor Adami's experience exactly coincides, so that in a well-marked case of leukoplakia one will nearly always find that there is a history of long-continued alcoholism. There is found also the form of œsophagitis in which the mucosa is dark red and granular, without any evidence of fibrin or membrane. All these varieties are of the same significance, and seem to occur under similar conditions.

Chronic œsophagitis may be characterized by a gray, œdematous mucosa as well as by a hyperæmic one; there is an increased quantity of mucus secreted; the thickening of the mucous layers occurs at times so irregularly as to give rise to papillate or polypoid outgrowths. As an accompaniment of this, the openings of the mucous glands are at times evident. Ulceration does not appear to be frequent, save in the most severe cases. Pigmentation of the mucosa is uncommon, and atrophy of the mucosa probably does not occur, save in old age.

Symptoms.—The most common complaint is uneasiness, scarcely amounting to pain, in swallowing; it is rarely severe, although this depends entirely on the degree of the lesion, and is generally referred by the patient to the upper part of the œsophagus, and by the observer to the pharynx, which is usually inflamed.

Sequelæ.—Because the infective agent is less virulent or the tissues more habituated, chronic inflammation is less likely to lead to severe destruction and subsequent stenosis than in the case of acute inflammation. It is likely that chronic œsophagitis precedes the unexplained cases of rupture and dilatation; it is, therefore, far more important in the light of those lesions to which it indirectly predisposes than for its own sake.

Treatment.—Various forms of treatment have been advocated, but the condition will continue to be, in most cases, untreated. The removal of an exciting cause, such as alcohol or tobacco, is necessary; extremely hot or pungent foods are to be avoided; it is possible by demulcent fluids to smear the œsophagus with material which will adhere to its walls for a time; the passage of a tube to apply such fluids or pastes is admissible. A useful method of applying astringent drugs to the œsophageal wall is the daily use of a soft tube smeared with a paste which is solid at ordinary temperature but melts at body heat. Such a paste can be made of cacao butter mixed with tannic acid (10 per cent.) or silver nitrate (5 per cent.). The tube is allowed to remain ten or more minutes until the paste is melted and transferred to the wall of the œsophagus. The condition is not sufficiently severe to compel serious measures, such as rectal feeding. Solutions of cocaine (1 to 2 per cent.) and eucaine (3 to 4 per cent.) are used to allay pain, and solutions of tannin and silver nitrate (1 per cent.) are advised. If such solutions are used, swallowing in the recumbent posture seems advisable.

Ulcers of the Œsophagus.—It is not exactly rational to separate these from the foregoing inflammations. Ulceration may occur as a step in the process of œsophagitis, but several forms of this lesion yet remain to be described. These are the so-called decubitus ulcers, pressure ulcers, peptic ulcers, and uræmic ulcers. Tuberculous ulcers will be dealt with in the paragraphs upon that disease. At the same time that these are separated from acute and chronic œsophagitis, it is done with no intention of suggesting that we are dealing with other than inflammatory processes, or at least those modifications of inflammatory processes which we are accustomed to call degenerations.

Decubitus Ulcers.—These are found in emaciated persons who have suffered from exhausting disease. They are usually shallow, round erosions, with little or no surrounding reaction, and are situated on the anterior and posterior walls of the œsophagus, at the level of the cricoid. Each is the counterpart of the other, and they are caused presumably by the attrition of the apposed walls while the patient lies recumbent, the œsophagus being pressed between the cartilages of the larynx and the vertebral column. They give rise to no symptoms.

Pressure Ulcers.—These may appear on the inner surface of the œsophagus as a result of compression from without by an aneurism or other mass. The lessened blood supply from the pressure doubtless lowers the resistance of the part, so that these are pathologically very closely related to the foregoing.

Peptic Ulcers.—So long as the etiology of gastric ulcer remains obscure, it is rational to consider peptic ulcer of the œsophagus as a sequence, although perhaps a distant one, of inflammatory conditions of the wall. It occurs generally in the lower part of the gullet under conditions similar to those in which gastric and duodenal ulcers occur, which, in fact, it often accompanies. Yet it must be said that it has generally been reported in elderly, alcoholic men, which fact alone casts doubt upon the exact parallelism. It may be said that the prevailing opinion is in favor of peptic ulcers being the result of a combination of thrombosis of the area affected with subsequent digestion.

Symptoms.—The condition causes difficulty in swallowing, and generally vomiting of blood, occasionally a fatal hemorrhage, and the course, apart from the last-named accident, is likely to be a chronic one. Diagnosis is

difficult and must be made from varices of the œsophagus and ulcer of the stomach.

Treatment.—This is exactly that of acute œsophagitis, gastrostomy being the most satisfactory method.

Uræmic Ulcers.—Ulcers of the œsophagus occur in uræmia, and are doubtless comparable to the better-known but equally ill-understood ulcerations found in the colon in uræmia; in thirteen cases of uræmia in the Royal Victoria Hospital, œsophageal ulcerations were found twice.

RUPTURE OF THE ŒSOPHAGUS.

Rupture of the œsophagus is rare, but has happened in a well-authenticated series of cases.

Etiology.—Much discussion has occurred as to whether a healthy œsophagus ever ruptures; the writer's belief is that it does not. At the same time, it must be stated that in some cases that have been carefully examined, no trace of disease has been found; with regard to this, it must be borne in mind that many degenerations are not evident to the examining eye, even in heart muscle, where we are accustomed to look for them.

"Spontaneous" rupture of the œsophagus has always occurred with a high internal pressure, such as is caused by the passage of a very large bulk of solid or fluid, coincident with a strong diaphragmatic contraction; thus, it is oftenest during severe retching or vomiting that the viscus gives way. Zenker considers that a rapid degeneration of the wall of the œsophagus is produced by the presence of a pepsin-containing fluid regurgitated from the stomach, which remains at body heat, at the same time that a condition of weakened œsophageal circulation is present; this degeneration is the predisposing, as the high intra-œsophageal pressure is the effective, cause of "spontaneous" rupture. Such degeneration could be effected only upon the internal layers, and it is extremely likely that an equally effective or more effective cause would be a degeneration, inflammatory, so-called, or other, of the muscular wall.

Symptoms.—Rupture of the œsophagus is characterized by severe, sudden pain in the chest, a choking sensation, retching, and collapse. All of the cases have proved rapidly fatal, although in some death has been sufficiently delayed to allow the signs of interstitial emphysema of the mediastinal tissue to develop.

IDIOPATHIC DILATATION OF THE ŒSOPHAGUS.

Idiopathic dilatation of the œsophagus will be dealt with when the subject of ectasia is taken up; suffice it to mention here that there are certain diffuse dilatations for which a cause cannot be assigned; in the absence of cardiospasm and other stenoses these are generally referred to as atonic degeneration of the wall. If it is really admitted that atony without any stenosis allows dilatation, it is reasonable to refer such atony to an unobserved, but nevertheless actual preceding chronic inflammation, or else to a muscular atrophy from malnutrition, which atrophy is so great as to allow a stretching of the weakened fibers until they are unable to recover themselves.

INFECTIOUS GRANULOMATA OF THE ŒSOPHAGUS.

Tuberculosis.—Tuberculous disease of the œsophagus is rare, but may occur by inoculation with tuberculous material which is swallowed; more commonly than this, the disease appears as areas of ulceration, which have extended from the bronchial lymph nodes, the trachea, occasionally the dorsal vertebræ, or even the lungs; or, finally, the œsophagus may share in a general miliary tuberculosis. Thus it will be seen that the œsophagus suffers late in the disease, when any symptoms to which it might give rise are overshadowed by others, so that the diagnosis can be made but rarely. There is no certain symptom or group of symptoms, although with advanced disease elsewhere severe pain on swallowing suggests its presence. Perforation of a tuberculous ulcer into the pleura has been reported (Flexner,¹ Stillman).

Syphilis.—Many authors deny that syphilis of the œsophagus occurs; this can scarcely be true, but the condition is certainly very rare. The cases described rest mainly upon therapeutic evidence, viz., that after antisypilitic treatment an apparent stenosis of the œsophagus had disappeared. One would suppose that the scars would remain, but no definite cases of such have been found in the literature. There is no reason to suppose, however, that the œsophagus is immune to syphilitic disease. Ulcers, also, have been described, and the morbid anatomy and treatment need no special comment, if one adds that of course antisypilitic treatment must accompany the local therapeutic measures.

Actinomycosis.—Actinomycosis and blastomycosis of the œsophagus have been described, and it may be here mentioned that trichiniasis also occurs.

NEUROSES OF THE ŒSOPHAGUS.

There is a tendency to think that neuroses exist without organic lesions, but in the œsophagus, as elsewhere, it is well to remember that some lesion may exist which is a point of origin for the stimuli that call forth the symptoms. The lesion may be, often is, quite inconsiderable and out of proportion to the symptoms it excites, but the results of treatment will at times be much better if it be borne in mind that such may exist. In many cases it must be admitted that not the slightest deviation from the normal state is to be detected.

Sensory Neuroses.—Disturbance of the sensory functions of the œsophagus usually takes the form of hyperæsthesia and paræsthesia; anæsthesia is said to occur after such infections as diphtheria, but is very difficult to determine. The two former conditions are more definite, and hyperæsthesia is frequently present, although it is often not a neurosis but an accompaniment of actual disease, such as inflammation or new-growth. Paræsthesia takes very different forms, such as pain in swallowing, a feeling as if the tube were closed, a sensation of weight, a feeling as if there were a foreign body in the œsophagus, and finally the sensation of a lump which is usually called the *globus hystericus*, which last is described at times as

¹ *Johns Hopkins Hospital Bulletin*, 1893, iv, p. 4.

ascending, and at others as descending. The surface of the œsophagus sometimes is described as feeling "rough;" in any of these conditions the distress may be less during the taking of food, although this is by no means to be depended upon, for the neuroses may be severe enough to lead to marked inanition by starvation. These paræsthetic states are often the accompaniments and sometimes the results of hyperacidity and allied conditions of the stomach that are called neuroses, and a familiar form is that described as "burning," which may extend as high as the pharynx; it must be kept in mind that this symptom is often the result of insufficiency of the cardiac orifice and the consequent regurgitation of (hyper) acid fluid. The use of the tube, sound, or œsophagoscope may demonstrate that there is no gross organic disease of the part, and the recognition of a neurosis of the stomach, or an unbalanced state of the nervous system in general, may be a useful guide to the recognition of the true state of affairs.

Diagnosis.—It is not easy to make useful suggestions as to the diagnosis of the neuroses, as the symptoms and the organic diseases that may be simulated are so numerous. In general, it may be said that the greatest care is necessary that organic disease be not overlooked, and the more unusual neuroses ought to be determined only after careful exclusion.

Treatment.—This requires that the general cause be recognized and the state of the nervous system in general be improved; because of the reality of the distress to the patient, symptomatic treatment is frequently necessary, and may take the form of regularly administered drinks, such as weak tea, soda water, weak solutions of tragacanth or acacia; in bad cases these may be followed by opium in small doses, tr. opii, $\text{m} \times$ (cc. 0.6), with aqua laurocerasi, 'xx (cc. 1.2) or 0.25 to 0.5 per cent. solution of silver nitrate, of which half a dram in water three times a day will usually suffice. Weak solutions of cocaine (1 to 2 per cent.) may be used by the help of the stomach tube and removed after ten minutes' application.

Motor Neuroses.—The motor neuroses are more important than the sensory, and are practically confined to spasm in various situations. A few cases of merycism, or rumination, do occur which are evidently connected with a relaxation of the cardiac orifice; some cases have been quoted to show that a real "fore-stomach" may occur as in ruminants, this hypothesis depending on the presence of a sac at the lower part of the œsophagus just above the cardia.

Before going on to speak of spasm proper of the œsophagus, it may be mentioned that some consider the globus hystericus to be spasm in its lowest degree; that is, that an actual point of contraction may pass up and down in a kind of peristaltic action of the tube. Whether this be the case or not, mild temporary spasms which last but for a moment do exist, and almost as soon as the patient recognizes that the food does not pass freely they yield, just as their better developed brethren yield to the application of the tube or sound. The patient has a momentary sensation that the food is obstructed, and that is all. It is only when the spasm is severe and consistent that there is damage wrought.

Etiology.—Spasm which is purely neurotic, called idiopathic, exists chiefly in young adults, rarely in children, and oftenest in females. Excitable, nervous people and hysterical persons are the ones most frequently afflicted, and a shock, fright, or anger is often the signal for an attack; the older writers seldom fail to mention the dread of hydrophobia as an excitant. Pregnancy

appears at times to have this disturbing effect. There are also cases which are not idiopathic but reflex, occurring generally in the same kind of people as the idiopathic. As a result of errant stimuli excited by a diseased condition of structures which have a related nerve supply, or stimuli arising in a functionally disordered central nervous system, or even stimuli originated by some slight lesion of the œsophagus itself, the motor nerves of the œsophagus carry impulses which result in a spasmodic contraction of the muscles, occurring most often at the upper part of the tube or at the cardiac orifice of the stomach. Notwithstanding the reality of the muscular contraction, the cases which have died with a spasmodic stenosis have failed to show any sign at autopsy, although one or two reported cases have indicated a possible persistence of the condition after death. The most usual site of a spasmodic closure of the œsophagus is at the cardia, and whether this be considered œsophageal or gastric matters not for practical purposes. These stenoses are found at other parts as well in the continuity of the tube, and the spasm is not necessarily always at the same place. When an organic stenosis exists, a spasmodic stenosis at the same or a different part of the tube may be superadded.

Symptoms.—The slighter degrees of the disease show a sudden, variable inability to swallow; one cannot predict what kind of food is likely to excite it; in some it is hot, in some cold, in some solid, and in some liquid; at times the larger the mass the more easily it is swallowed. The attempts at swallowing are often audible, and the attention of others is apt to increase the difficulty experienced by the patient. Variability of symptoms is very noticeable. Emaciation is absent in the slight or moderate cases. In severe cases dysphagia may be complete, so that regurgitation of material eaten some time before may happen, the material indicating by the absence of hydrochloric acid or pepsin that it has not reached the stomach. The act of attempting to swallow is accompanied by dragging pain in the retrosternal region, and fluids are sometimes more difficult to swallow than solids. Dyspnoea, oppression, palpitation of the heart, syncope, and even convulsions may accompany the attack. The condition may be momentary or continue for hours, and the liability to recurrence has been known to last through half a long lifetime.

Diagnosis.—The diagnosis is important, and must be made from inflammatory narrowing, stenosis by malignant growth, general or partial dilatations, impacted foreign bodies, or even from the pressure of a mass outside the œsophagus. A goitre has been known to exert such pressure. To distinguish spasm from diverticulum, it must be remembered that in the former there can be no real ructus or vomiting of stomach contents during the spasm, but the tube alone can be emptied (Meltzer); in diverticulum the stomach may regurgitate food, although the diverticulum be full. The fluoroscope is essential for such observations. The age, sex, and character of the patient are of importance, and the most useful single characteristic of spasm of the œsophagus is the variability of the symptoms. Very complete information may be obtained from the use of the stomach tube, which is sometimes found to be grasped by the stricture, most often at the cardia; after a few moments or minutes the spasm passes off, and the tube passes easily; if a sound be used it ought to be of fairly large calibre, such as 1 cm. diameter, but information is at times subsequently obtained by the different diameters of the sounds that will pass on different occasions in the same patient. The

sound is generally the quicker, and from the physician's standpoint more satisfactory than the tube. It must not be forgotten that organic lesion may co-exist with spasmodic stenosis. If large sounds do not pass, it is rational to give an anæsthetic, and if it pass during anæsthesia, the condition is spasmodic. Stenosis which disappears after the use of antispasmodics, the sound, or galvanism can be almost certainly determined as spasmodic.

Treatment.—In this it is obvious that, as in other neuroses, the main object is to render the general health and tone of the nervous system so good that the spasm will cease to occur; if a local cause exist, to which the spasm may be attributed, it should be removed; such causes have been found in chronic disease of the tonsils, the presence of foreign body in the ear, intestinal parasites, and many diverse slight derangements to which attention would scarcely be directed in time of health. The food which the patient thinks the best is desirable, although the character of this is subject to change. Generally, warm or sweet fluids are less apt to excite spasm than cold or acid ones.

In cases that are at all severe the use of the tube or sound is necessary, and it is often notable that although the use of the sound will usually overcome the spasm, it will not necessarily prevent its recurrence; when the spasm has yielded to the sound or the tube, it does not always follow that food taken immediately will be allowed to pass, but such is generally the case. The tube, or preferably the sound, should be inserted gently until it meets the obstruction, where it is sometimes grasped; if grasped, it should be allowed to remain until released, which generally happens in a few moments or minutes; if not grasped, a gentle equal pressure should be kept up until the obstruction is felt to yield. If the spasm be frequent, it is of the very greatest importance that the patient's nutrition be kept up; in severe cases, rectal feeding is useful, and even the performance of a gastrostomy for temporary use is at times justified.

OTHER ŒSOPHAGEAL AFFECTIONS ARISING FROM THE NERVES.

Paralysis.—This is known as a sequence of diphtheria, and is characterized by partial or complete inability to swallow, the first indication of its existence frequently being the regurgitation of liquids through the nose, due to an associated paralysis of the palate. In an uncomplicated paralysis of the œsophagus the food bolus sticks, and the gullet is as little able to regurgitate it as to pass it downward. As soon as it is recognized, the greatest care must be exercised lest food be aspirated into the air passages. If the physician is in doubt as to the efficiency of the muscles of deglutition, tube feeding must be instituted. The condition is curable only by time, although the constant hypodermic injection of strychnine, gr. $\frac{1}{20}$ (gm. 0.003) thrice daily, appears to be of advantage.

Herpes.—At least one well-authenticated case of herpetic eruption¹ in the œsophagus has been reported, but it is worthy of mention rather as a curiosity than as a condition likely to be met, as is also pemphigus,² which may be mentioned as an actual happening, although its insertion under the present chapter heading is scarcely justifiable.

¹ Holub, *Ther. der Gegenwart*, September, 1906.

² Tamerl, *Wien. klin. Woch.* 1904, No. 29.

DILATATION OF THE ŒSOPHAGUS.

Diffuse dilatation or ectasia of the œsophagus is that form of widening which occurs generally above a stenosis of the lumen, although it may occur when no stenosis is evident. It is generally fusiform, although the weight of the ingesta tends to make a considerable bulging toward the lower part of the sac; in the case of a stricture, spasmodic or organic, the greatest dilatation usually occurs immediately above the site. The majority of all ectasias are in the lower third of the tube.

Etiology.—Although sometimes found as a congenital lesion, an organic stricture of the cardia or other part of the tube is the most common cause; it is generally believed that spasm of the cardia can give rise to the dilatation, which is brought about by the pressure of the food which is unable to make its way through the orifice. Many so-called idiopathic cases¹ have been described, upon the etiology of which it is idle to speculate; it is supposed in these cases, as well as in those which are secondary to stricture, that atony or degeneration of the muscle has occurred. In some of the cases of this class a marked hypertrophy of the muscle is found, but this is more likely to be a resultant and compensatory change subsequent to the formation of the ectasia than a primary condition. Fleiner, Strauss, and others consider that these dilatations are explained by a congenital defect in the enteromeres, and that this is combined with a cardiospasm of neurogenic origin. Those spindle-shaped ectasias which are widest at the middle are probably due to the muscular weakness being greatest at that part, but the shape as finally observed at autopsy is probably quite different from the shape that is assumed when food is passing down; at such a time it is likely that the most extreme dilatation exists in the lowest part of the ectasia. Beneke's² view is a sensible one, which is that the traction and bulging of the lowest part of the sac, when it contains food, pulls together the walls of the upper part of the undilated tube below, thus causing a kind of valve action at that point, which still further prevents the egress of the sac contents. The wall of the œsophagus in a dilatation is generally inflamed, and often ulcerated, a condition which assists greatly in keeping up the causative spasm. With respect to the occurrence of dilatation above a stenosis, the statement is made that it does not often occur, but personal observation has not borne out this view.

Symptoms.—These are generally masked by those of the stricture which gives rise to the dilatation; there is difficulty in swallowing, fluids and soft foods being most easily taken; the saliva is increased; regurgitation of undigested food with mucus is common; when the dilatation contains food there is often pain in the sternal region, a sense of oppression, dyspnœa, and regurgitation of the contents of the sac; with decomposition of the ingesta, foul breath and a bad taste in the mouth are present. The sound may pass without difficulty, although sometimes it will be obstructed in so decided a way as to give rise to the supposition that an organic stricture exists, even in a spasmodic case; when it is in the dilatation, the excursion of its distal end is

¹ Pietkrowski, *Arch. f. Verdauungskrank.*, Band x, Heft 2. See also Lerche, *American Journal of the Medical Sciences*, 1907, cxxxiv.

² *Deuts. Aerzte Zeitung*, June 15, 1901.

freer than usual. In ectasia, as in diverticulum, the sac may be emptied and the contents found to lack hydrochloric acid, pepsin, and rennin, or the tube cannot be inserted as far as the stomach. The frequency with which the wall of the sac becomes inflamed by the decomposition of food brings it about that there may be continual discomfort in the sternal region, even when there is no food present in the œsophagus.

Diagnosis.—This depends upon careful observation of the symptoms and examination of the contents of the sac for the absence of stomach secretions; the use of the tube or sound may demonstrate the wide sac and the causative stenosis, and sometimes the spasm. When the condition is suspected, it can best be verified by the use of a bismuth mixture put into the œsophagus and viewed by the fluoroscope, although Kraus points out that in early cases even this, as a rule, fails. The conditions with which it is most likely to be confused are organic stenoses without dilatation and deep diverticula.

Prognosis.—Ectasia, if not extreme, is consistent with long life, although occasionally death is brought about by inanition, which should be considered rather as a result of the stricture than the dilatation. The prognosis in an uncomplicated case rests upon the completeness with which nutrition can be carried on. The presence of severe inflammation of the wall is a menace, in that the muscle may be affected and weakened and the dilatation thereby increase, but especially because ulceration may occur, which carries with it the danger of perforation.

Treatment.—In cases due to spasmodic or organic stricture the treatment of the primary condition dominates the case. Treatment of the dilatation is unsatisfactory. When it is recognized as arising from cardiospasm, combined with treatment of the spasm, electrization of the walls by a stomach-tube electrode has proved beneficial.

Apart from the treatment of the organic stenosis or the spasm, it is often useful to wash out the sac every night, and so avoid the decomposition of its contents, and combat the inflammation by the use of silver nitrate solution (1 to 3 per cent.) or solution of boracic acid (2 to 3 per cent.). The treatment of the causative stenosis and cardiospasm is dealt with under those heads.

DIVERTICULA OF THE ŒSOPHAGUS.

Etiology and Pathology.—Diverticula of the œsophagus are pouches of the wall whose cavities connect with the lumen of the viscus; the sac may be connected with the organ by a narrow neck, or it may be funnel-shaped, in which case no neck, properly so-called, exists. Diverticula are classed as "pressure diverticula" when they are caused by pressure from within, and "traction diverticula" when the force is exerted from without. These two forms have nothing in common, save that both are dilations of the œsophageal wall, yet custom has led to their being treated together. Their pathology is widely different, and pressure diverticula, which will be treated of first, are most frequently not œsophageal but pharyngeal diverticula. Of pressure diverticula, then, it may be said that they are at times congenital, but the majority are the result of disease in the œsophagus or in the surrounding structures. The congenital diverticula are usually due to defective fusion of the muscle coats of the two sides at the median line

posteriorly, and when lateral appear to result from a lack of complete obliteration of the branchial clefts. Most diverticula, however, are not of this nature, but result from diseases in the œsophageal wall.

Pressure diverticula¹ are oftenest found high up on the *posterior* wall of the œsophagus at the point where pharynx and œsophagus meet, opposite the unyielding cricoid cartilage, but they may be found elsewhere, when they show a predilection for the natural narrowings of the tube. Their causation is doubtless due to the fact that the effect of pressure of the wall between a firm bolus of food and a hard organ externally (such as tracheal calcification, a calcified thyroid, or even, it is said, a calcareous artery) is to injure the tissues; they are deprived of physiological rest by the constant movements entailed in eating and drinking. When the œsophageal wall is thus weakened, the muscular and supportive tissues become insufficient and stretch easily; after each considerable distention the wall fails to contract to its proper degree, and the lumen of the tube remains a little wider at this point than normal, and each successive dilatation increases the size of the pouch. When the wall is weakened, a diverticulum is caused by a stretching of the muscle fibers and a separation of one from another; when an individual group of fibers break, or when this separation occurs, a hernial protrusion of the mucosa and submucosa through the gap follows; rarely these protrusions are still covered by an incomplete muscle layer, but even when this is not the case the thickened submucosa forms a wall that is at times as thick as the normal œsophagus. When they result from such a hernial protrusion, they often have a narrow, slit-like opening; notwithstanding this, they may attain a large size, when their liability to rupture constitutes a real menace to the safety of the individual. The increase of intra-œsophageal pressure by the passage of food distends yet more the beginning sac, and because of their production thus by pressure from within, these are called "pressure or pulsion diverticula." They occur most frequently in the male sex, and in middle or advanced life; it is supposed by some good authorities that pressure diverticula occurring in the continuity of the tube are really the final result of traction diverticula; if this be true, these would be properly called "pressure traction diverticula," a class described by some authors, which is for our purposes a needless refinement.

The "traction diverticula" are produced in quite a different way from that already described; some nearby structure, such as a lymph gland, gains adhesion by inflammation to the outside of the tube, and the contraction of the inflammatory tissue during the process of fibrosis or scar formation makes a constant traction on the wall, which is pulled out at this point. A high, intra-œsophageal pressure during the passage of food assists this process, and a small, frequently funnel-shaped diverticulum results. Since the lymph nodes at the tracheal bifurcation are oftenest the cause of this form of diverticulum, it is most frequently found at this level on the *anterior* wall. Tuberculosis, extremely common in these glands, is a frequent excitant; Zenker and Heller have pointed out their liability to inflammation secondary to pericarditis; caries of the spine has also caused it. Ritter declares that chalicosis is a more frequent cause than tuberculosis. While this is the most commonly accepted view on the causation of traction diverticula, Ribbert² and others hold that when the air passage is separated first

¹ See Riebold, *Deuts. Archiv f. klin. Med.*, 1904, lxxx.

² *Virchow's Archiv*, 1904, Band clxxviii.

from the œsophagus, a band of connective tissue is left, connecting the tracheal and œsophageal walls; the œsophageal muscular wall is often not perfect at this point (B. Fischer),¹ and the result of a traction by this band, which traction may be caused by any slight displacement of the tube, is the formation of a diverticulum; some have thought that the spot at which an artery enters is thereby weakened and suitable for dilatation, but the anatomical sites of traction diverticula argue against this view. Schmorl found traction diverticula in 3.5 per cent. of all autopsies² (a figure which appears excessive to an American observer), and one-third of these had led to perforation into surrounding tissues, under which circumstances they may lead to infiltration and subsequent gangrene of the lungs, rupture of a blood-vessel, or some such untoward accident. These diverticula appear so insignificant on the œsophageal wall that one cannot at first understand that they are liable to perforation; yet one in our own series appeared to be only 2 or 3 mm. deep, but a fine probe inserted passed up what looked like a fibrous cord, fully a centimeter to the peritracheal gland situated on the outside. In this narrow passage a small piece of solid material, such as a seed, might readily become caught, and the funnel shape of the diverticulum tends to allow any such foreign body to pass up into the narrow channel beyond. Once lodged here, it could ulcerate its way out. Before leaving this subject it may be said that the question of the origin of diverticula of the œsophagus has of late years occupied attention far beyond its merits and the volume of controversial literature on the subject is very large.

Symptoms.—Traction diverticula, from their small size, give no symptoms and are rarely discovered during life, and then only by œsophagoscopic examination. Pressure diverticula of the œsophagus, if small, give no symptoms and are frequently not recognized. If, however, a diverticulum be large, and be filled by liquid or food material, the decomposition of this and the irritation produced thereby will give rise to vomiting; the patient may feel a sensation of "something in the throat;" there often is difficulty in swallowing, which is greater when the diverticulum is full, so that it compresses the œsophagus proper. Under such circumstances respiration may be interfered with and the neck may be larger than usual, or an actual tumor appear. Pain is rarely present, unless ulceration of the diverticulum exists; it must be remembered that even a small diverticulum may become the seat of ulceration by lodgement of a seed or small, hard food particle.

From the decomposition of its contents foul breath arises, and the inflammation so set up may spread to the œsophagus itself, or may even proceed to ulceration and rupture, with the formation of peri-œsophageal abscess; or the break may occur into the thoracic cavity, setting up gangrene of the lung or empyema; a diverticulum has been known to burst into a tuberculous cavity of the lung (Schmidt). When the diverticulum is small the tube will readily pass it, but as it becomes larger the tube is often directed into the sac itself, where the mobility of its lower end and the impossibility of passing it farther may assist the diagnosis. The tube is especially liable to pass into the sac if the latter is full, thereby pushing the œsophagus forward out of the direct line and taking its place.

Diagnosis.—This may be made occasionally by palpation if the diverticulum be sufficiently high to appear in the neck; if filled with air it will

¹ *Virchow's Archiv*, 1904, No. 178.

² *Riebold, Deut. Archiv f. klin. Med.*, 1904, lxxx, Heft 5 und 6.

be tympanitic, if with fluid, dull on percussion, and external pressure will sometimes succeed in emptying it. If lower down, the failure to find hydrochloric acid in the regurgitated material may indicate that it does not come from the stomach, and the stomach tube may succeed in reaching fluid at so high a level that it could not be in the stomach; if the tube can be passed with certainty into the diverticulum, which is often a matter of much difficulty, a mixture of carbonate of bismuth and syrup of acacia can be introduced, which will render the diverticulum opaque to the *x*-rays. It must always be borne in mind that the sac is easy to perforate. The diagnosis has been made by recognizing food which has been eaten several meals previously, when the stomach has previously regurgitated food more lately taken. However, unless one or other of these accidental circumstances give a clue, the recognition of diverticulum is very difficult.

Treatment.—Diverticula large enough to be recognized demand treatment, although treatment is not very easy. Feeding by the tube, when it is possible to pass it by the orifice of the diverticulum, is advisable, and if the shape of the sac be determined, and the neck of it found to admit the procedure, surgical removal should be advised. This as yet applies only to those situated in the neck, but the development of the low-pressure chamber of Sauerbruch and others for thoracic operations promises to extend the possibilities of operation. In the event of a diverticulum being recognized, and the advent of ulceration observed, *e. g.*, by the presence of blood in the contents, rectal feeding will be necessary for a time because of the danger of rupture. While erosions are healing, potassium bromide, ʒij (gm. viii), or morphine, gr. $\frac{1}{2}$ (gm. 0.03), daily, may be used to lessen irritability, although physiological rest by withholding food is the most rational procedure. Regular washing with water greatly assists the patient by the prevention of decomposition, thus lessening inflammation, and by increasing the tone of the muscle. At intervals of a few days, a solution of silver nitrate (1 per cent.) may be used, and the washing of the sac can be taught to an intelligent patient. When cardiospasm is present, the occasional passage of a large bougie will assist the overcoming of it, but this procedure should always be done by the physician and never by the patient. Electrolysis is widely used by employing a gastric electrode inside the sac. The most important requirement is a well-regulated rich diet; fats, butter, and oils are desirable, and when ordinary feeding is practised, one or two teaspoonfuls of melted butter or olive oil are recommended to be taken a little before food time, to act as a lubricant. Many patients are able to empty the sac by certain gymnastics, such as the motions of rowing; it is obvious that, apart from washing, a sac can be scarcely kept clean by such measures alone.

STENOSIS AND STRICTURE OF THE ŒSOPHAGUS OF NON-MALIGNANT ORIGIN.

Obstruction of this nature, apart from benign tumors, to be spoken of later, may be congenital, spasmodic, or fibrous. The spasmodic variety has been already dealt with as a neurosis, but it clinically deserves mention under the above heading, because a given case may be difficult of differentiation for a long time.

PLATE VI

FIG. 1



FIG. 2



FIG. 3



Fig. 1.—Stenosis of lower half of oesophagus of a child, resulting from drinking lye. Dilatation of gullet above the stenosis.

Fig. 2.—Epithelioma of oesophagus. A flat, shallow, ulcerated growth is seen spreading over a relatively large surface.

Fig. 3.—Epithelioma of oesophagus. The upper part of the growth is flat, the lower much more nodular. The stomach wall is partly cut away to show a lymph node with great secondary involvement.

(From specimens in McGill Medical Museum.)

Etiology.—A congenital stenosis has been undoubtedly found in a number of cases with no trace of fibrosis; it is generally in the upper part of the œsophagus, annular or fusiform in shape, and concerns the mucosa and submucosa only. A case is described by Mayer which underwent cure. The condition is an extremely rare one,¹ and before deciding that a stenosis is congenital the history should be carefully searched for even a slight trauma.

The fibrous stenoses take origin in any inflammation, wound, corrosion, burn, or ulceration in which the loss of substance is so great that any considerable degree of fibrosis replaces it. In a general sense it follows that the greater the loss of substance, the more certainly will a serious degree of stenosis occur, and the greater the length of surface implicated in the original lesion, the greater will be the longitudinal extent of the stenosis. In the case of corrosives, the more concentrated they are, the greater likelihood there will be of a rapidly ensuing stricture, while much diluted corrosives are most apt to form more slowly developing and less complete stenoses. Thus it follows that with a very severe corrosion the patient will probably never be free from dysphagia until the stricture is formed, whereas the lesser grades of corrosion may seem to heal, and after a period of normal patency one finds with disappointment that the sequelæ of the corrosion have to be reckoned with, months later, in the guise of a slow stenosis. The sites where stricture is most likely to occur are the natural narrowings, *i. e.*, at the cricoid, the tracheal bifurcation, and the diaphragm, yet definitely annular stenoses are at times found elsewhere. The peculiar liability of these parts is due to the momentary delay of the corrosive or burning material at these points. The entire œsophagus has been stenosed in certain cases. When the stenosis is once established a moderate degree of dilatation is often to be observed above it. (See Plate VI, Fig. 1.)

Sequelæ.—In cases of stenosis, inflammation, either due to the injury which caused the stenosis or as a secondary result of it, may spread to the surrounding structures, or may lead to perforation; in a case not thus complicated there is little likelihood of disturbance of the surrounding tissues; so much is this the case that the existence of pressure on the recurrent laryngeal nerve is strong evidence against stricture. A possible sequel is injury or even perforation by the sound, especially when dilatation or pouching of the œsophagus above may allow the sound to stray from the lumen.

Symptoms.—Not to deal with the symptoms of the causative injury, the invariable sign of stenosis is difficulty in swallowing, which is constant, generally progressive, and fluids are more easily taken than solids. Pain is generally absent. The food that does not pass through is regurgitated after a variable time; the presence of a dilatation above the stricture is likely to be accompanied by delay in the regurgitation. The patient generally knows his capacity for swallowing liquids at any time, and will, in partial stenoses, not exceed the amount that can pass down; the food or drink is felt to "stick" at a certain point, referable generally to the top of the sternum. The point where the stenosis is can be determined accurately by the tube or sound, although a second lower stenosis may exist; when the site of the stenosis is determined, graduated sounds should be used, beginning with the large and proceeding to the smaller ones, until one is found which can enter the stricture. It is to be remembered that the existence of the stenosis

¹ See Thomas, *Lancet*, February 6, 1904

tends to excite spasm, and this at first may make the stenosis appear more extreme than it really is. If the stenosis is at all considerable, the auscultatory sounds can be recognized as ceasing at the affected level. In the absence of treatment, any degree of stenosis will be attended with loss of weight from malnutrition, because the mental anxiety attendant on the dysphagia tends to keep the patient from using his œsophagus even up to its limited capacity.

Diagnosis.—The diagnosis must be made between this condition and stenosis from carcinoma or from spasm; foreign bodies (which have been known to remain in the œsophagus for astonishingly long periods), polyps, and external tumors pressing on the œsophagus must also be considered. The history of injury is of the highest value. In any case, before proceeding to the examination, one must be reasonably sure that aneurism does not exist; then one must consider carcinoma, because it is the most frequent and likely cause; its rapid progress and the greater degree of emaciation, with absence of a definite trauma, are all useful indications. The *x*-rays will frequently determine if a foreign body be present; the benign tumors, such as polyp, are rare, but they may be impossible to differentiate, save by the observation of the non-progressive nature of the lesion.

Prognosis.—This cannot be given in so many words, but depends upon the degree to which nutrition can be kept up and the certainty with which aspiration of food into the respiratory tract can be avoided. The earlier the stage at which treatment is begun, and the more youthful the patient, the better is the prognosis for cure.

Treatment.—Treatment is palliative and curative, and the latter should always be tried. Many surgical procedures have been advised which depend for their reasonableness upon the site of the obstruction; excision of the stricture, internal and external œsophagotomy, even the cutting of the stenosis by fishing-line, have all been, at times, successful; the last method has something mediæval about it, but is advised by some, especially after preliminary gastrostomy. Rapid dilatation is dangerous, but in cases of cardiac stenosis gastrostomy followed by dilatation from the stomach is rational. Considered broadly, the best treatment is gradual dilatation, which is performed by the passage of the largest sound that will pass, every other day for several times, then using the next largest similarly for a time; the sound should be left in for ten or more minutes at each treatment. In this way the stricture will sometimes yield completely, after which it is well to pass a large sound at rare intervals to be warned of a recurrence and to prevent it. If nutrition is failing, gastrostomy should be done, even while the treatment is in progress, or before it is begun. A most important fact to keep in mind is this, that careless use of the sound may irritate the surface and add to the stimulus that is already producing the fibrosis, while the danger of perforation, especially when there is dilatation of the gullet above the stenosis, must always be remembered. Fortunately, the stenosis is generally funnel-shaped, and, therefore, the sound is insensibly guided in the right direction.

Various other methods of dilatation have been devised, such as rubber-bags, which are distended to any degree required, being attached to a gauge. Laminaria tent sounds have also been used, but the gum-elastic and the whalebone sounds with ivory tips are still the favorite method. A number of case reports, chiefly from German sources, speak favorably of the employ-

ment in organic stricture of thiosinamin (allyl sulphocarbamide); repeated injections are made hypodermically of gr. i to ij (gm. 0.06 to 0.12) every two or three days, and the result is said to be softening of the stricture to such an extent that where bougies could not be passed they were readily admitted after a few administrations.

Palliative treatment includes such measures as gastrostomy and the wearing of a permanent tube to allow of passage of fluids through the stricture; the latter device is especially useful, as it eliminates the spasmodic increase of the stenosis. This procedure will be treated of under the heading of Carcinoma of the Œsophagus. As a palliative measure, œsophagostomy is not as simple as gastrostomy, and is probably not preferable.

CARCINOMA OF THE ŒSOPHAGUS.

The most important disease of the œsophagus is cancer, which commands attention by reason of the readiness with which the narrow lumen of the organ is obstructed, as well as by the importance of the neighboring structures which are likely to be infiltrated. So frequent is the occurrence of carcinoma that we may say that in every patient between the ages of forty and seventy years, with a stenosis of the œsophagus without other evident cause, the odds are overwhelmingly great that it is carcinoma.

Cancer of the œsophagus is surpassed in frequency of occurrence only by cancer of the uterus, female breast, and the stomach. Figures, as usual, are conflicting; our Montreal series of 3882 autopsies shows 24 cases of œsophageal carcinoma, a percentage of 0.61. Kraus¹ gives figures which aggregate 216 cases out of 45,405 autopsies, a percentage of less than 0.5; but as some of these series date back as far as 1830, it is possible that earlier cases may have been overlooked. The Russian statistics show an astonishing frequency of the disease, one series indicating that the œsophagus was the seat in more than 32 per cent. of all carcinoma cases; a series of 981 cases of carcinoma, compiled from Kraus, gives a percentage of 20 for the œsophagus, but this contains three series of Russian cases. Our series at the Montreal General and Royal Victoria Hospitals, in 3882 cases, gave 265 carcinomas, 24 of which were of the œsophagus, *i. e.*, 9 per cent. In this series it is surpassed in frequency by carcinoma of the stomach, female breast, and of the colon; into this list, ordinarily, would come the uterus, although, as usually happens in General Hospitals, autopsy figures in regard to carcinoma of the uterus are very low. In the last report of St. George's Hospital, London, the œsophagus ranks second only to the stomach in frequency. In using autopsy figures it must be remembered that the breast and uterus, which are comparatively accessible and admit of surgical treatment, show a frequency of occurrence far below their real position.

Etiology.—Carcinoma of the œsophagus is a disease of middle or advanced life, being found most frequently between the ages of fifty and sixty years; the earliest case of which a record was found was at nineteen years; one case in our own series occurred in a woman aged thirty-two years. The male is much more liable than the female sex, the largest series

¹ Kraus, *Die Erkrankungen der Speiseröhre*, Wien, 1903 (Nothnagel's *System*).

showing a ratio of 3 to 1. Smaller series show as great a preponderance as 7 to 1.

Our knowledge of the causative factors of this disease, as in carcinoma elsewhere, is very small; irritation has been considered as of more moment than any other contributing cause, and alcoholics are considered as specially liable. Stress is laid upon the occurrence at the physiological narrowings, and upon the histories frequently given by patients in whom the first symptoms followed the swallowing of a mouthful of hard food or of hot material. The arguments in favor of this as yet appear insufficient. It is also to be pointed out that carcinoma not infrequently seems to spring from scars of the œsophagus, from traction diverticula, and from the walls of dilatations; but this, as well as its occurrence in drinkers, is probably but another way of saying that chronic inflammatory conditions predispose to its occurrence. Paul Wolf¹ cites three cases, to which Schmorl adds two, where carcinoma of the œsophagus developed at the site of a spondylitic deformity of the vertebræ. He points out that it may be that the vertebral deformity caused a bend in the œsophagus at this point, so that irritation caused by the passage of food was added.

Special Pathology.—Carcinoma of the œsophagus is nearly always primary, and its most common mode of occurrence as a secondary growth is when it spreads by extension from a growth of the cardiac end of the stomach, or less frequently from the thyroid gland, in which cases the growth is of the same nature as the original neoplasm. In the Royal Victoria Hospital series, while 12 primary cancers occurred, only 3 secondary ones were found.

The tumors vary much in appearance and extent, perhaps because one sees them at autopsy in so widely different stages. By reason of the organ involved, death frequently occurs from intercurrent disease or accident, and lack of nutrition is always prominent. Thus it happens that often one is enabled to see a growth not long after its commencement. At times the carcinoma appears as an isolated mass on one wall, oftenest the anterior; at other times a flat growth spreads over a relatively great area (Plate VI, Fig. 2). Gray or white or yellowish white in color, it often shows little scattered islets extending from the periphery of the main growth; very frequently the growth is annular, encircling the tube (Plate VI, Fig. 3). Again, a section through the stenosis will show a yellowish-white thickening of the wall of the gullet with comparatively slight evidence of nodular growth on the mucosal surface; or again, one finds a large, soft, irregular, cauliflower-like, fungating mass that seems to more than fill the lumen of the œsophagus, whose ulcerated surfaces bear witness to the difficulty with which patency of the tube has been maintained. As a rule, not more than one or two inches of the tube are implicated, but cases have been known to affect the entire length of the organ. All degrees of hardness are found, but generally the mass is soft; a real scirrhous carcinoma has not come under personal observation. Degenerations are the rule, and necrosis of large parts of the tumor is very commonly seen. The consistence obviously depends on the relative amount of cellular tissue of the tumor and stroma, and in addition any degeneration will render it yet softer. One is frequently struck by the fact that the tumor mass seems inconsiderable in comparison with the symptoms to which it gives rise.

¹ *Münch. med. Woch.*, 1903, No. 18.

The degree of stenosis depends on the size and the consistence of the growth plus the possible spasmodic stenosis set up by its presence. By the lodgement of food and the weakening of the walls a dilatation is often formed above the stenosis. It is generally considered that dilatation is apt to occur because the growth is rapid and the wall constantly stretched by food without there being sufficient time to allow an effectual hypertrophy. Variations in the degree of stenosis occur, so that the occurrence of ulceration and the detachment of pieces may be the signal for the subsequent easier passage of food. Ulceration in the main growth is almost sure to occur from the attrition of the apposing walls of the gullet, and from the passage of food; the irregularity of the growth makes many small hollows and bays, in which decomposition is likely to be set up and the ulceration thereby hastened. Strangely enough serious hemorrhage from such ulcerations is rare, and the amount of blood is small; pieces of the growth which are detached either are regurgitated or pass into the stomach and are digested.

Histologically, most œsophageal carcinomas are squamous-celled (epitheliomata), although adenocarcinomas are found in a certain number of cases, taking origin from the glands scattered throughout the organ. Of the former class, many are properly basal-celled carcinomas: perhaps one might say that all of the squamous-celled carcinomas exhibit somewhere in their structure the basal-celled quality. It always seems to the writer that the difference between these two closely related histological forms is merely a difference in the grade of reversion (kataplasia or anaplasia) of the epithelial cells giving rise to the tumor, a difference which often, but not always, is closely correlated to the activity of growth and multiplication of the same. The growth is frequently seen arranged in the familiar circular masses, and true epithelial "pearls" are generally to be observed; various differences are seen in the amount of fibrous stroma that exists, and the more fibrous stroma there is, the less is the malignancy of the growth, or perhaps it is better stated that the more fibrous stroma there is, the less rapid is the progression of the growth. Mucoid degeneration has been described, and a single report of primary carcinoma myxomatodes is to hand (O. Fischer¹).

Site.—The commonest site of œsophageal carcinoma has been a matter of endless debate and is very difficult to designate. The level of the bifurcation of the trachea seems to be affected oftener than any other place, but as to which third of the gullet is the most liable to carcinoma, a study of the literature indicates the lowest third; but this statement is made without conviction; it does not greatly matter, for it is a well-proved fact that no part enjoys any exemption. A favorite text-book statement is that the points of physiological narrowing are more liable to be attacked than are other parts, but this statement loses value when one finds that, according to Kraus, thirteen different points of narrowing have been described in the 25 cm. length of the tube. Few disputed points can be so safely left unsettled as this one. It does seem possible to state, however, that a very small fraction, perhaps a tenth of all growths, lie in the neck, where they are surgically accessible.

Perforation.—This is surprisingly frequent; in Petri's series of 44 cases, 27 perforated. The perforation may occur into different organs or areas,

¹ *Prag. med. Woch.*, 1899, xxiv, 30, 31.

depending upon the site of the growth, but in by far the greatest number the ulceration is into the larger air passages, the larynx, trachea, or a bronchus; apart from these, into the mediastinal tissue, the pleura, the lung, or even, although a rarity, into the pericardium. Perforation is not of itself necessarily fatal, but death follows soon, as a rule, because the œsophageal contents set up aspiration pneumonia or gangrene. When the rupture occurs into the mediastinum, interstitial emphysema of the tissues of the neck may be seen, to be followed by cellulitis. When the pleura is densely adherent to the lung, the lung tissue itself may be directly invaded by the material escaping from the opening. Occasionally the perforation occurs into a blood-vessel, as will be detailed below.

Extension to Neighboring Structures.—Many different structures may be invaded and damaged by the extension of the growth, chief among which are nearby arteries, the vagus or the sympathetic nerves, the recurrent laryngeal nerve, if the growth be left-sided, or even the thoracic duct and the brachial plexus. The vertebræ have been eroded, and an œsophageal carcinoma has been known to produce pressure on the spinal cord. In the case of the veins, the gradual pressure of the invading neoplasm leads to a thrombosis, so that by the time the wall is eroded the danger of hemorrhage is obviated. In the arteries, on the other hand, there may be no such protection, and fatal hemorrhage has occurred in a number of cases. Most of the important arteries near the œsophagus have been observed to be implicated by extension of œsophageal cancer; the carotids, the subelavians, the intercostal and vertebral arteries have been invaded, and of 50 cases of arterial implication collected by Knaut, 32 occurred in the thoracic aorta. Those cases in which the heart, generally the auricles, has been attacked by the growth, where particles have been broken off and carried to other parts of the body, are to be regarded rather as curiosities than as actual clinical probabilities. A growth at the cardiac end is very apt to spread to the peritoneum or into the diaphragm.

For many years an impression prevailed that carcinoma of the œsophagus had not so great a tendency to form metastases as had carcinomas elsewhere, but this arises from no inherent peculiarity of the growth itself; it possibly happened that the secondary growths in the deep cervical glands were often overlooked; it is even more likely that the importance of the structures involved leads to a comparatively early fatal result, at which time the lymphatic involvement, and especially extensive involvement of distant organs, is as yet not prominent. The comparatively short duration of carcinoma of the œsophagus, compared with that of other sites, is in favor of this explanation. Twelve cases at autopsy in the Royal Victoria series showed secondary growths in 8.

Symptoms.—In the majority of cases the earliest symptom is a difficulty in swallowing, which at first occurs only with a hard or dry mouthful of food, with a feeling of discomfort at the site of the growth; as the patient describes it, there is "something there." A mouthful of fluid generally suffices at first to wash down the obstructed mass. In one case of our own series the stenosis at death was so great that the circumference of the gullet at the growth was 1.5 cm., yet the patient, having been living on fluids, never complained of dysphagia. The progress of the difficulty in swallowing is at times slow, but is constant; an exception to this is that sometimes late in the disease a piece of the growth may be broken away and the lumen

thereby widened. At first, merely hard pieces of food give difficulty or discomfort, then there is obstruction to soft foods, and finally to fluids, so that ultimately none or at best very little fluid can be taken at a time. Attempts at swallowing excite cough, and frequently regurgitation occurs, which may be immediately after the food is taken, or delayed for a considerable time, even hours. The higher up the stenosis, the more prompt is the regurgitation, and the lower, the longer delayed. Regurgitation of food is frequently preceded by severe retching, and the regurgitated food is generally alkaline, mixed with much mucus, and shows no peptonization. If the growth be ulcerated, the regurgitated material may be mixed with blood, and occasionally particles of the growth may be found; under these circumstances the breath is usually fetid.

Commonly, discomfort or a feeling of oppression is felt under the sternum, localized to the site of the growth, although severe pain may be complained of; it is at times referred to the xiphoid region, instead of to the actual situation of the growth. The severe pains are of a tearing or piercing character, at times radiating widely to the back, neck, or shoulders. Such pains are often nocturnal, and may be independent of attempts to take food; although pain is, in most cases, a late symptom, it must not be forgotten that it may be the earliest indication of the disease, preceding even the difficulty in swallowing.

With the gradual increase in the difficulty of taking food there is great weakness; the loss of flesh is rapid, and may be extreme, while many patients complain of severe thirst, less frequently of hunger. The cervical and supraclavicular lymph nodes, when involved by secondary growth, are enlarged and at times palpable, especially on the left side. It is scarcely necessary to give in detail the symptoms that arise from pressure upon the various nerves which may be implicated. It is said that in as many as a sixth of all cases there is a contraction of the corresponding pupil, with ptosis, falling in of the bulbus, and sluggish reaction to light (Hitzig); these pupillary changes are most often seen on the left side, and depend upon pressure on the sympathetic nerves; there need not be with it a paralysis of structures supplied by the recurrent laryngeal. The involvement of the sympathetic means that lymph nodes have implicated the nerves, and does not tell the site in the œsophagus; especially is involvement of the right recurrent apt to be by lymph nodes, because on the left side the nerve lies so close that the primary tumor may affect it. When the recurrences are involved, there may be a very serious dyspnoea, which may come on even before dysphagia has become a prominent feature. More common than any other single symptom, probably, is paresis or paralysis of the vocal cords from interference with the recurrent laryngeal nerve.

As soon as difficulty in swallowing has called the attention of the physician to the possibility of stenosis of the œsophagus, provided that no contra-indication exist (the presence of aneurism should always be excluded), a local examination by the use of the tube and bougies must follow; a stomach tube of 1 cm., or greater, diameter is to be used, and it can thus be determined that an obstruction really exists, and its exact position can be recognized. If bleeding is excited by the use of the tube, the further examination by bougies should be postponed, but should no such contra-indication exist, one can sometimes determine, by using bougies of diminishing calibre, at what level in the growth the stenosis exists, and may

even guess at its extent. The greatest care should be used in passing the bougie to avoid perforating the wall, an occurrence by no means infrequent in careless hands. When using the tube, the contents of the eye of the tube should be noted, as pus, blood, or mucus may be found; more rarely, particles of the growth are obtained. The examination of the contents of the stomach is not of moment, for normal findings, hyperacidity or anacidity may exist.

Of great value, but of some difficulty of application, is the use of the œsophagoscope, by which the growth can be actually seen, and its nature and extent, at least as far as the stenosis, be accurately determined. An early diagnosis can be made with sureness by no other means, and therefore, when it is possible, it should be used. Its employment by the physician has as yet obtained little or no foothold in America, but in most hospitals it is possible to make use of its information. For the diagnosis of an advanced case it is rarely necessary.

Diagnosis.—This is well summed up as follows: "If there is steady loss of flesh, with increasing dysphagia, in a middle-aged or elderly person, suspect carcinoma; if the sound is arrested, the diagnosis is very probable; if there is slight bleeding with the use of the sound, it is all but certain." In its early stages the disease may be confounded with almost any œsophageal disease; it may preserve its likeness to benign stricture for a considerable time; an advanced case is generally recognized without difficulty. It is obvious how useful œsophagoscopic examination by a trained eye may be in an early case.

Prognosis.—The duration of the disease varies between six months and two years from the appearance of the first symptoms, although at times its entire course is measured in weeks. The prognosis must be admitted to be very bad; carcinoma of the œsophagus in an accessible part is but a small proportion of the whole, and even in these surgical treatment must admit a large mortality and a small percentage of cures. Death occurs most frequently from inanition and cachexia, while next in order of frequency come the results of perforation into and implication of the respiratory tract.

Treatment.—Treatment is in most cases palliative, but in a few may be curative. The few cases in which the growth is early and accessible offer chances of success under surgical treatment, and even the intrathoracic growths are now being attempted. In all cases other than the above the treatment can be only palliative; the principles of such treatment are simple; keep up the nutrition, feed by the mouth as long as is possible, counsel an œsophagostomy or gastrostomy, preferably the latter, when mouth feeding is impossible or about to become so, and keep the patient as free as may be from pain.

The preservation of nutrition is the most important factor, although difficult of accomplishment. Good judgment and the utilization of the patient's experience are requisite in the selection of articles of diet. The stringy mucus which so often troubles the patient is lessened by the use of solutions of sodium bicarbonate (1 to 10) as a wash for the mouth, or to be swallowed. A demulcent quality in the food is desirable.

Many physicians practise intubation of the stricture, for which various procedures have been devised. One of these is a tube, of which the upper end is fixed in the back of the mouth (by cords fastened around the ear or in other ways); it passes through the stricture to the stomach. Another method is the use of a short tube, with a projecting rim which rests on the

stricture, which passes the narrowing. It is placed in position by a whale-bone guide, and is also secured above by cords through the mouth. Of these, it seems as if the form which gives the less friction is the more desirable. Care of the cords is necessary. If a fistula into the respiratory tract has occurred, the use of a tube is essential to the continuance of mouth feeding, and at this point it may be stated that most clinicians are of the opinion that it is necessary to carry on mouth feeding as long as possible, to utilize, for one thing, to the full degree, the salivary secretions and their admixture with the food.

With the gradual increase of the stenosis it becomes evident that further means need to be undertaken, lest the patient die rapidly from starvation; œsophagostomy below the stricture or gastrostomy is indicated, and for various reasons gastrostomy is the operation generally preferred. It is a difficult matter to counsel a gastrostomy at the right time; it is almost certain to be required, and if delayed too long the patient is apt to die from the effects of the operation, or the degree of starvation is so great that its good effects are not realized. On the other hand, the patient will often object to the operation so long as the œsophagus remains moderately patent. No rule can be laid down for a decision; a frank explanation of the circumstances to the patient and the patient's friends should be made.

If pain be severe, it should be treated. The application of a few drops of weak silver nitrate solution (1 or 2 per cent.), or 10 to 15 drops of a 3 to 4 per cent. solution of eucaine, or 10 drops of a 1 per cent. solution of heroin, repeated thrice a day, will be found to relieve pain; 10 drops of a 1 per cent. solution of the extract of *nux vomica* taken thrice daily is recommended; the astringent solutions of silver nitrate will at least assist cleanliness; if these prove inefficient, the hypodermic injection of morphine is the most satisfactory method.

The external use of the *x*-rays has not been of service, but Einhorn¹ reports benefit in retardation of the growth from the use of a radium vial in a hard-rubber capsule, which is screwed into the end of a flexible rubber tube. The vial is left in position for one-half to one hour at a time.

SARCOMA OF THE ŒSOPHAGUS.

Sarcoma of the œsophagus occurs rarely, and cannot be differentiated during life from carcinoma, save by the microscopic examination of fragments of the growth. Its treatment is identical with that of the commoner neoplasm.

NON-MALIGNANT GROWTHS OF THE ŒSOPHAGUS.

Non-malignant new-growths of the œsophagus deserve only brief mention. The commonest is fibroma, or fibrous polyp, which not infrequently is pharyngeal in origin, and as it grows hangs down into the œsophagus. Rokitsansky reported one seven and a half inches in length and two and a half inches in thickness. An interesting case (Monro), coming from the Edinburgh Hospital in 1763, appeared in the Edinburgh Physical and Literary

¹ *Journal of the American Medical Association*, July 1, 1905, xlv.

Essays of that time. By movements of vomiting and coughing it could be projected to the front teeth, but had to be swallowed almost immediately to prevent choking. A ligature was tied around it, it was re-swallowed, and allowed to slough, the piece coming away per anum. Two years later it caused the death of the patient.

These growths may be sessile or pedunculated, the former being probably the earlier stage; they are elastic, firm, and are apt to be ulcerated; although generally single, they may be multiple. Myomata, fibromyomata, and lipomata¹ also occur; finally, more frequently than any of the last named, are those proliferations of mucosa and submucosa called papillomata, which are pedunculated, soft, and often multiple. They rarely give rise to symptoms of any degree of severity. All the signs of new-growth may fail with these neoplasms, provided they be not large enough to cause difficulty in swallowing, but if they are large, one may find, as in carcinoma, pain in the chest radiating to the back and epigastrium, becoming accentuated on swallowing, especially if ulceration be present; the differential diagnosis rests upon the preservation of nutrition if actual sight of the tumor by the cesophagoscope is not possible. Surgical treatment may become necessary.

¹ See Glinski, *Virchow's Archiv*, 1902, Band 167.

CHAPTER IV.

FUNCTIONAL DISEASES OF THE STOMACH.

By JULIUS FRIEDENWALD, M.D.

GASTRIC NEUROSES.

Introduction.—Gastric neuroses are those functional disturbances of the stomach manifesting symptoms of a nervous character and not based upon any anatomical pathological changes. A certain number of the so-called gastric neuroses, dependent without question upon some form of anatomical change, have up to the present time eluded our means of detection. On the other hand a number of the diseases that were formerly classified as neuroses are now known to be accompanied at times by certain anatomical changes, and cannot therefore be properly classified simply as neuroses. Thus, on the one hand, the condition of hyperchlorhydria has been shown to be frequently accompanied by a proliferation of the cells in certain portions of the mucosa, while, on the other hand, degeneration of the gland cells has been found in a large proportion of cases of achylia gastrica. With more and more exact methods of investigation, the number of gastric neuroses will gradually be lessened in number, and some of these diseases be placed upon a more definite anatomical basis.

It is now but little understood in what manner the disturbances of innervation take place; it is well known, however, that the branches of the vagus and abdominal sympathetic carry the impulses to and from the stomach, and it is through these nerves that these disturbances of innervation occur; while we cannot trace the exact paths, it is generally believed that the impulses of motility, sensation, and secretion are transmitted by different nerves; since any of these disturbances may exist alone and independently of the other, we therefore believe in the existence of special nerves of motion, secretion, and sensation.

It will not be out of place to briefly discuss a few points associated with the nervous mechanism of the stomach. Between the circular and longitudinal muscles there is a plexus of nerves (Auerbach's plexus) by means of which a local nervous mechanism is maintained. Intimate connection with the great nerve centres is had through the vagi, while in addition to these the sympathetic sends fibers to the stomach. It has not yet been entirely determined whether the rhythmic contractions of the stomach and intestines are entirely under nervous control, since this special process is regarded by some as being of muscular origin alone. Magnus¹ considered these contractions of nervous origin. By experiment he succeeded in obtaining rhythmic movements of the muscles of the intestines taken from the body,

¹ *Die gesammte Physiologie*, 1904, cii, p. 362.

which still held the plexus of Auerbach, even after the removal of the plexus of Meissner and the mucosa.

It is still doubtful whether or not the pylorus is partially under local nervous control, for Cannon¹ has shown that destruction of either of the vagi or the sympathetic system does not prevent the passage of the chyme from the stomach into the intestines. There is no question, however, that the sphincters of the stomach are partially controlled by the central nervous system.² With special reference to the cardia, Langley³ has shown that the vagus contains both inhibitory and accelerator fibers, while Openchowski⁴ has demonstrated that stimulation of the sympathetic increases the contraction of the cardia. In reference to the pylorus, May⁵ has shown that the stimulation of the vagus causes at first an inhibition followed by an acceleration of the contraction of the pylorus. In regard to the nervous mechanism of other portions of the stomach, May has demonstrated that stimulation of the vagus caused at first an inhibition of the peristalsis of the pyloric portion, with a lessened tone of the cardia, followed later by an acceleration of peristalsis in the pyloric region with increased tone in the cardia. Cannon⁶ has shown that emotion plays an important role in the movements of the stomach by demonstrating that peristalsis ceases whenever "the animal manifests signs of rage, distress, or even anxiety." According to Cannon⁷ the stomach is divided into two parts that are physiologically distinct. The larger part is the cardiac area, the smaller the pyloric. During the process of digestion there are constant peristaltic movements running over the surface of the pylorus. Peristaltic waves are not present in the cardiac area. After the pylorus empties itself, however, the cardiac portion produces a tonic pressure, forcing its contents into the pyloric area; therefore, mixture with the gastric secretion takes place in the pyloric area. The food remains, however, for some time in the cardiac area, during which the salivary digestion is continued. Cannon has demonstrated that the stomach does not empty itself at once, but gradually and intermittently passes on its contents during the period of digestion. According to this investigator the presence of free hydrochloric acid in the stomach occasions the relaxation of the pyloric sphincter, causing the pylorus to open and so permit the escape of the acid chyme into the duodenum. The presence of the acid in the duodenum produces a reflex stimulation of the pylorus, which closes and remains so until the contents of the duodenum have again become alkaline.

Etiology.—Gastric neuroses may be primary or secondary; they are primary when the seat of the nervous disorder is inherent in the stomach itself, and secondary when the nervous mechanism of the stomach is reflexly affected either from the brain, spinal cord, or some other organ, such as the liver, kidneys, etc. Thus, a severe pain, such as kidney colic, may reflexly affect the stomach, causing vomiting; it is also a well-known fact that menstrual disorders as well as sexual disorders in males may have a marked reflex effect upon the gastric functions. It has been shown by Gould that⁸ severe eye strains may also have a marked influence in this regard. On the

¹ *American Journal of Physiology*, 1905, xiii, p. 22.

² *Medical News*, May 20, 1905.

³ *Journal of Physiology*, 1898, xxiii, p. 407.

⁴ *Centralblatt für Physiologie*, 1889, S. 1.

⁵ *Journal of Physiology*, 1904, xxx.

⁶ *American Journal of Physiology*, 1898.

⁷ *Ibid.*, 1904.

⁸ *Journal of the American Medical Association*, March 24, 1906.

other hand, gastric neuroses may in turn reflexly affect other organs, and we not uncommonly find such symptoms as headaches, palpitation, insomnia, depression, and other manifestations as a result.

Occurrence.—The writer has classified the cases of gastric neuroses occurring in his practice during a period of some years; in order to obtain a clearer view of these cases, the histories have been collected both from the records of private cases and hospital practice. This plan was deemed advisable because comparatively few forms of gastric neuroses are seen in the hospital wards. Of 2000 patients suffering with the various forms of gastric disease, 1592 (79.6 per cent.) represented one or more forms of nervous disorder, while 408 (20.4 per cent.) were affected with organic disease.

In order that gastric neurosis may exist, there must be present some predisposition, *i. e.*, a neurotic condition in the individual. This is frequently first manifested by nervous symptoms directed to other organs, the stomach only later becoming involved either gradually or suddenly. In this series of cases the early nervous manifestations were first noted in 38 per cent. in organs other than the stomach, while in 62 per cent. the stomach itself was first involved. The manner of development of these affections is often peculiar, at times beginning suddenly, often without any apparent cause or error in diet, persisting for a longer or shorter period of time, and then perhaps terminating abruptly, at other times coming on slowly, progressing rapidly, and terminating when least expected. Heredity plays an important role in the development of gastric neuroses. Many persons inherit a susceptible nervous system, while others develop these conditions by improper modes of living, bad habits, excesses, cares, and trials; in consequence of this an unstable nervous equilibrium is produced from which the nervous gastric disorder takes its origin. Among these observations 70 per cent. of the patients gave a history of either some functional or some organic hereditary nervous tendency. In 32 per cent. the disorder was directly dependent upon some shock, anxiety, or vice, while in 18 per cent. no known cause could be discovered.

Most of these affections are more frequently observed in females than in males, more often in men of the higher classes, and about equally in females of the lower and higher classes. In this series of cases 54 per cent. represents the number of females affected, while 46 per cent. indicates the number of males. The chief factor in the production of these conditions in males is overwork, worry, and excesses, especially overindulgence in drink, 29 per cent. of the patients giving a distinct alcoholic history. The chief causes in women are disorders of menstruation and reproduction, anxiety, sorrow, and disappointments. The predominance in this sex is due to the mode of living and peculiar affections.

Age.—The neuroses rarely begin in old age. They occur most frequently between the twentieth and fortieth year. The following table shows the number and percentage of gastric neuroses occurring at various years in this series:

Year.	Cases.	Per cent.
1 to 20	232	14.5
20 to 30	354	22.5
30 to 40	404	25.0
40 to 50	339	21.5
50 to 60	175	11.0
60 to 70	88	5.5

The neuroses occur as frequently in the robust and well-nourished as in the broken-down and enfeebled individual. After persisting for some time neuroses interfere with the general health, frequently occasioning emaciation; this condition, however, is secondary in most instances and not a predisposing factor in the etiology. The symptoms of a general neurosis are usually present, *i. e.*, irritability, lassitude, insomnia, depression, and a feeling of malaise. Hyperæsthesia or anæsthesia often exist in certain parts of the body. The subjective symptoms are changeable and capricious, exhibiting protean changes in rapid succession. The digestion is usually, as Boas states, in a condition of "labile gastro-intestinal function." The digestive complaint is frequently independent of the quality and quantity of food ingested, and without any connection with its digestion; errors of diet are not necessarily followed by aggravation of the symptoms. Periodic attacks of discomfort often alternate with unaccountable periods of well-being. Frequently sudden changes take place in the secretory or motor functions of the stomach, or in both, so that a superacidity may quickly give way to a subacidity and a motor insufficiency to a hypermotility. The pain, also, which may be present is diffuse and often bears no relation to the digestion of food.

Gastric neuroses are usually polysymptomatic, more rarely monosymptomatic in character. In the first form there is a multiplicity of symptoms, while in the latter but one symptom is observed. Neuroses rarely present the same form during their entire period, and a monosymptomatic neurosis is not infrequently converted into the polysymptomatic form. A characteristic feature of gastric neuroses is the fact that the symptoms vary so frequently, which is often of great assistance in differentiating between this class of troubles and actual organic disease.

Classification of Gastric Neuroses.—The following classification is the one most frequently adopted in the majority of text-books:

I. Secretory:

- (a) Subacidity or hypochlorhydria.
- (b) Anacidity or achylia gastrica.
- (c) Superacidity or hyperchlorhydria.
- (d) Supersecretion or gastrosuccorrhœa. Continua periodica (Reichman). Continua chronica.

II. Sensory:

- (a) Bulimia.
- (b) Akoria.
- (c) Parorexia.
- (d) Polyphagia.
- (e) Hyperæsthesia.
- (f) Sitophobia.
- (g) Gastralgia.
- (h) Gastralgokenosis.

III. Motor:

- (a) Rumination (merycism).
- (b) Regurgitation.
- (c) Incontinence of pylorus.
- (d) Eruetatio nervosa.
- (e) Pneumatosis.
- (f) Peristaltic unrest.
- (g) Vomitus nervosus.
- (h) Cardiospasm.
- (i) Pylorospasm.
- (j) Atony.

Nervous dyspepsia (neurasthenia gastrica).

This classification has by no means been found to be satisfactory, and Boas¹ has suggested the following modification: He divides the gastric neuroses into two large classes—monosymptomatic and polysymptomatic. In the monosymptomatic variety one symptom is prominent or alone observed, and may be either of a depressive or of an irritative nature:

MONOSYMPOMATIC GASTRIC NEUROSES.

Irritative Group.

Superacidity or hyperchlorhydria.
 Supersecretion or gastrosuccorrhœa.
 Gastromyxorrhœa.
 Bulimia.
 Parorexia.
 Gastralgokenosis.
 Gastralgia.
 Hyperæsthesia.
 Rumination (mercism).
 Regurgitation.
 Eructatio nervosa.
 Vomitus nervosus.
 Nausea nervosa.
 Cardiospasm.
 Pylorospasm.
 Pneumatosis.
 Peristaltic unrest.

Depressive Group.

Subacidity or hypochlorhydria.
 Anacidity or achylia gastrica.
 Akoria.
 Anorexia.
 Sitophobia.
 Incontinence of pylorus.
 Atony.

POLYSYMPOMATIC GASTRIC NEUROSES.

Nervous dyspepsia.

Diagnosis.—This may be very difficult, as frequently these conditions envelop some organic disease, the nervous symptoms being so much more prominent that the actual disease becomes entirely masked. In order to establish the nervous character of a gastric disorder, all organic diseases must be excluded, and this is frequently a most difficult task. In the monosymptomatic forms the diagnosis is usually simple, especially if the examination of the gastric contents and functions shows a normal condition. The patient frequently has a hereditary nervous tendency or a weakened nervous system or neurotic habit. The disease is frequently either characterized by peculiar periodical or paroxysmal attacks, with many unaccountable periods of well-being, or by an absence of subjective symptoms even upon the ingestion of indigestible food.

The diagnosis of the polysymptomatic forms is more difficult. In these the symptoms that are frequently found in severe organic diseases may be interwoven and combined with the gastric neuroses; a further difficulty arises from the fact that organic disorders are often accompanied by nervous symptoms, and a most careful investigation may become necessary to clear up the diagnosis.

Nervous dyspepsia is the most typical example of the polysymptomatic neuroses of the stomach with a multiplicity of symptoms seldom found in its frequency in organic diseases of the stomach. According to Boas² the nervous dyspeptic himself will often indicate the diagnosis of this condition,

¹ *Deut. med. Wochenschrift*, August 17, 1906.

² *Ibid.*

and one need only listen to his story attentively. Objective signs may be absent, or, when present, may mislead one; thus a slight dislocation of the kidney or a slight gastric atony would be insufficient to account for the symptoms present. In the diagnosis of these conditions the appearance of the attacks without apparent cause and the intervals of well-being are most striking. The fact that as a rule the complaints of the patient bear no relationship to the quantity and quality of food digested, but are mainly dependent upon overexertion, mental disturbances, and excitement is of greater value in the diagnosis. The conditions are often relieved or improved by change of scene or relaxation.

When it is impossible to gain a clear idea of the condition, Boas advises the functional testing of the stomach. The patient is given a very digestible diet, such as is usually prescribed during the third week of an ulcer treatment. The subjective symptoms are noted while the patient is on the diet, during a period of from three to four days; then additions are made gradually, such as sauces, vegetables, dessert, and raw fruits. An exact record is again made, and by comparing the symptoms during the first and second period the presence of an organic or nervous gastric disorder will be indicated. In the latter condition the symptoms not only often diminish, but frequently disappear entirely during the second period, while in the former they are apt to increase. Boas also advises the frequent testing of the motor and secretory functions of the stomach, which when found to be constantly normal are often alone sufficient evidence to indicate the neurotic nature of the disorder.

However, it not infrequently happens that even in nervous conditions disturbances of secretion and motility occur, demonstrating that after all, in many of these conditions, the diagnosis is best determined by the careful observation of the experienced diagnostician rather than by any of the more or less complicated methods of investigation.

Prognosis.—The prognosis in neuroses of the stomach is very favorable, provided the condition is treated early and the cause discovered and removed. Relapses, however, are frequent if care is not taken to regulate the daily regimen and maintain so far as possible a stable condition of the general nervous system. It is not uncommon, when relapses do occur, that some nervous manifestation is produced other than that which was originally present. In the series of 1592 cases there were 752 (47 per cent.) that gave a history of one or more relapses; of these, 321 (20 per cent.) had one and 431 (27 per cent.) more than one relapse; in the remaining 840 no evidence of any former attacks could be obtained. Many of the patients with relapses gave evidence that indicated previous neurotic conditions at some time other than those associated with the stomach.

Treatment.—In this it is necessary to remember that one is dealing with patients whose imagination is easily influenced in directions other than normal; for this reason the personal influence of the physician himself will have much to do with the patient's recovery. It is at first always important to make a most careful and complete examination of the patient to convince him that there is no organic disease present. A change of scene is often important, and specific rules as to the mode of living and diet should be insisted on. In some cases a sanatorium treatment is most desirable. The diet should be carefully regulated, and especially in badly nourished patients the aim should be to strengthen the individual and add to his nutrition by increasing the body weight. For this purpose also the physical methods of

treatment are most serviceable and must be resorted to, acting at the same time as suggestive measures. Of these, massage, cold and warm douches, packs, poultices, intragastric as well as external electricity, douching and lavage of the stomach play a most important role.

Sadger¹ extols hydropathic treatment for these conditions, and adapts the treatment to the constitutional weakness of the patient rather than to special symptoms. The treatment in this direction must be tonic and stimulant. He advises cold sponging with rubbing, cold compresses, the half or full cold bath, the Scottish douche, and Winternitz's compress. It is best in most instances to begin with mild forms of treatment, as the nervous dyspeptic is usually broken down and emaciated, making the applications more severe as the patient becomes more accustomed to them. The procedure is as follows: Early in the morning a limited sponging with water is given in bed at a temperature of from 50° to 54° F. If the patient is weak he is required to lie in bed with the window open for half an hour afterward; otherwise he is immediately sent into the open air. After a few days, depending upon the reaction to the treatment, the above process is replaced by rubbing of the entire body for three minutes with water at from 57° to 60° F. Besides the rubbings, one or two sitz baths are given daily at a temperature of 60° to 50° F., for ten to fifteen minutes, and moist abdominal bandages are applied every three to four hours. If the moist compress becomes unbearable, it should be preceded by energetic rubbing of the abdomen with the hand moistened in cold water. If this is insufficient, a reaction is first induced by placing a hot-water bag upon the abdomen, over a moist cloth, and following it by the cold compresses. Following this treatment cold douches are indicated, the tendency being not only to apply treatment to the abdominal organs, but to strengthen the entire nervous system.

The Winternitz compresses consist of a coil of rubber tubing between a dry and moist layer of cloth through which water flows at a temperature of 104° F. for from one to two hours. They are especially useful in cases of nervous dyspepsia associated with nausea, vomiting, and pain after eating. By means of this application patients are enabled to eat and retain nourishment that otherwise could not be given. When sanatorium treatment is impossible or impracticable, a sojourn at the seashore or mountains, with specific directions as to the mode of living, rest, exercise, and diet, often proves serviceable. A cure at some mineral spring is often beneficial, provided the proper spring is selected and specific directions given as to drinking the water and the mode of living to be followed. In no instances should patients suffering with nervous stomach troubles be permitted to tell others of their condition. In severe forms a rest cure should be resorted to, as most remarkable results can often be achieved by this means.

The nervous dyspeptic should be taught to rely more upon proper hygienic measures than upon drugs, as but few drugs have any marked influence in these cases. Of the remedies that may be resorted to at times, the bromides, preparations of valerian, arsenic, and codeine are most useful.

HYPERACIDITY OR HYPERCHLORHYDRIA.

By the term hyperacidity is meant an excessive flow of gastric juice or, more correctly, an increase in the secretion of the hydrochloric acid produced

¹ *Archiv. f. Verdauungskr.*, Bd. xii, Heft i.

by the gastric mucous membrane during the process of digestion. In this condition the pepsin and rennet ferments are also increased in amount. It has been questioned whether hyperchlorhydria should be termed a distinct disease, inasmuch as the excess of acid is simply a symptom found in a number of gastric disorders and due to a variety of causes; yet the symptoms associated with this condition are so definite and so often embrace all subjective manifestations which can be elicited in a large proportion of cases that we are forced to treat this condition as a special clinical entity. This fact becomes the more apparent when we take into consideration that, although hyperchlorhydria is in most instances a secretory neurosis, in a certain number of cases it is associated with some definite pathological lesion (Hemmeter,¹ Straus²).

The question as to what is meant by the excess of hydrochloric acid has not yet been thoroughly agreed upon by all writers. According to Ewald, Einhorn, and others the normal percentage of free hydrochloric acid one hour after one Ewald test breakfast ranges between 0.1 and 0.2 per cent. Any amount above 0.2 per cent. is to be considered a hyperacidity; on the other hand, many cases are met with having a greater acidity than 0.2 per cent. without symptoms of hyperacidity. Thus, Kaufmann³ found in 19 patients who were free from gastric symptoms, 10 who presented a total acidity above 70 and 2 above 100. Geigel and Blass⁴ report 8 cases presenting no gastric symptoms whatever, and yet showing an acidity between 60 and 95. Stockton, Meyer, Illoway, and Steele have reported similar cases. In 14 of the patients in this series with free hydrochloric acid ranging between 0.21 and 0.29 per cent., and a total acidity varying from 70 to 100, there were no evidences of any gastric disturbance whatever.

On the other hand, we have already pointed out that symptoms of hyperacidity may exist even in cases of hypochlorhydria. This has occurred in 11 of the series. In the largest proportion of cases, however, any increase above 0.2 per cent. free hydrochloric acid represents a superacidity, and is usually accompanied by symptoms of this condition, although this is not always the case, for there are patients with acidities as high as 0.29 per cent. without any symptoms whatever, and others in whom free hydrochloric acid is entirely absent in whom symptoms of hyperacidity are manifested. The very same amount may indicate a hyperacidity in one individual and not in another, showing that individual variations may exist in the normal percentage, and that we cannot always draw a sharp line of demarcation between the normal and the hyperacid state. The condition has been explained in two ways; according to some writers, as Illoway,⁵ the stomach of each individual has its own degree of acidity, and thus may perform its function with an acidity which is insufficient or more than sufficient for another. Therefore, any increase into this normal proportion of acid will render this particular gastric secretion superacid for this individual and occasion symptoms of hyperchlorhydria. Another explanation which seems to be more satisfactory was suggested by Talma.⁶ According to this observer the symptoms of hyperchlorhydria are due in these cases to a hyper-

¹ *Diseases of the Stomach*, third edition, p. 814.

² *Virchow's Archiv*, 1898, Bd. cliv.

³ *Zeitschrift f. klin. Med.*, vol. lvii, p. 491.

⁵ *New York Medical Journal*, May 25 and June 29, 1907.

⁶ *Zeitschrift f. klin. Med.*, 1884, Bd. xiii.

⁴ *Ibid.*, vol. xx, p. 233.

sensitiveness or hyperæsthesia of the gastric mucosa to hydrochloric acid. Stockton¹ pointed out that the gastric mucosa is not only often intolerant to an excess of hydrochloric acid, but often to a normal or subnormal percentage of acid. Steele² confirmed the observation, and went a step farther to show that the hyperacid condition of the gastric contents is not alone capable of producing the symptoms in hydrochlorhydria; according to him there must be some other condition present occasioning the gastric hyperæsthesia, producing that condition in which the gastric mucosa cannot tolerate a normal or abnormal amount of acid, and, further, this hyperæsthesia is due to ulcer, retention or hypersecretion on the one hand, or to a sensory neurosis on the other. Kaufmann³ also believes that some other factor must be involved to occasion the symptoms of hyperacidity besides merely the increased amount of the secretion. According to him, whenever the patient presents the symptoms of hyperacidity a second pathological factor must be sought, forming the connecting causal link between the chemical findings and the subjective disturbances. In many of these cases this factor is demonstrated in some ulceration, erosion, dilatation, and by far most frequently in an atony of the stomach.

According to him, when these disturbances were cured and the atony relieved, in every instance, no matter whether the acid secretion had been reduced or not, the symptoms disappeared. In another series of cases no anatomical lesion or atony could be discovered, such cases having been proved to be purely neuroses, and all were cured by appropriate treatment when the neurasthenia was relieved.

Etiology.—Hyperchlorhydria is the most frequent of all gastric disturbances. In the series of 2000 cases, representing organic as well as functional disturbances of the stomach, there are 1273 (63 per cent.) showing a hyperacidity; in the series of 1592 cases of gastric neuroses there are 542 cases of hyperchlorhydria, or 34 per cent.

The disease is frequently observed in young and middle-aged persons, while it is rarer in older individuals. It is found to a slight degree more frequently in females than males. The following table indicates the number of cases in males and females at various ages:

Years.	Males.	Females.
10 to 20	34	18
20 to 30	54	59
30 to 40	84	96
40 to 50	52	71
50 to 60	22	21
60 to 70	17	14
	<hr/> 263	<hr/> 279

Hyperchlorhydria is found more frequently among the better and wealthier classes than among the poor. This is due to the fact that mental strain and worry are often direct etiological factors in the production of this disorder. Jaworski pointed out its frequency among the Polish Jews; we have observed it as a most frequent condition among the Hebrews of this vicinity. The following etiological factors in its production may be noted:

¹ *Journal of the American Medical Association*, January 11, 1903.

² *Ibid.*, August 18, 1906.

³ *Zeitschrift f. klin. Med.*, vol. lvii, p. 491.

1. Mental strain, mental overwork, prolonged worry, and mental fatigue are important. Nervous individuals and persons who have undergone severe mental strain are especially prone to this disorder; Van Noorden has found it frequently in patients suffering with melancholia. Neurasthenia and hysteria are also important causative factors; of the 542 cases of hyperchlorhydria; 186 belong to this class.

2. Gastro-intestinal atony is a marked etiological factor, especially those forms associated with chronic constipation. Ebstein,¹ Illoway,² and others have called attention to this fact. It occurred in 164 of this series, mainly in young females.

This condition is explained by Illoway in the following manner: Long-continued constipation is associated with gastro-intestinal atony, on account of which the gastric contents are retained for an unduly long period of time, and thus cause an irritation of the gastric mucosa, in consequence of which more gastric juice, with an increased acid secretion, is liberated.

The following is a typical example of this condition: T. R., female, aged twenty-one years, who had been suffering from chronic constipation for five years, complained of no gastric discomfort until the past six months, when the chronic constipation became more aggravated, even heroic doses of purgatives often proving ineffectual; heartburn, acid eructations, regurgitation, and gastric pains were present. The physical examination of the abdomen was negative. The gastric contents showed a total acidity of 84 free hydrochloric acid (0.22 per cent.). The use of large oil enemata retained overnight, in addition to a moderately regulated diet, relieved the constipation as well as all of the other symptoms within a few weeks, notwithstanding the fact that no other drug was used.

3. Indiscretions in diet, such as the use of food of a very heavy character, as well as the abuse of alcoholic drinks and tobacco, are the cause of the largest proportion of cases of hyperchlorhydria. This condition is frequently found in persons who masticate their food imperfectly, who eat too quickly, who drink foods too hot or too cold, or take their foods too highly seasoned. This class is represented by 186 cases in this series.

4. Among other conditions that bear some etiological relationship are ulcer of the stomach, chlorosis, cholelithiasis, and nephrolithiasis. It is not our province to discuss the connection between ulcer and hyperchlorhydria here. Oswald³ pointed out the association of hyperacidity with chlorosis; in 85 per cent. of his cases a hyperchlorhydria existed. In 34 cases of this series with chlorosis and gastric disturbances, hyperacidity was found in 24, normal acidity in 8, and subacidity in 2. The following table represents the cases of chlorosis in relation to the degree of acidity.

Cases.	Total acidity.	Per cent. of free HCl.	Per cent.
2	10 to 40	0 to 0.1	6
8	40 to 60	0.1 to 0.2	24
24	60 to 110	0.2 to 0.35	70

That cholelithiasis is frequently accompanied by a hyperacidity has been pointed out by Kaufmann⁴ as well as by Friedenwald.⁵ Any condition leading to biliary retention may produce this condition.

¹ *Deut. med. Wochenschrift*, 1904, p. 1749.

² *New York Medical Journal*, May 25, June 29, 1901.

³ *Münch. med. Wochenschrift*, 1894, Nrs. 27, 28.

⁴ *American Medicine*, 1903, vol. vi, No. 20.

⁵ *Medical News*, July 22, 1905.

The following table represents the cases of hyperchlorhydria, tabulated according to age and sex, presenting the various etiological factors already enumerated:

Causes.	No.	Males.	Females.	Ages in years.		
				10-30.	30-50.	50-70.
1. Mental overwork and fatigue, neurasthenia	156	89	67	41	71	41
2. Gastro-intestinal atony with chronic constipation	164	54	110	73	54	37
3. Indiscretions in diet, food, drink, and tobacco	186	105	81	95	57	34
4. Indefinite and unknown causes	36	15	21	—	—	—

Pathology.—As hyperchlorhydria is largely a sensory neurosis, no anatomical lesion is present in the greater proportion of cases. Oesterreich¹ found the gastric mucous membrane perfectly normal with the exception of a few slight erosions in a patient suffering from hyperchlorhydria dying of an intercurrent pneumonia.

According to Hemmeter,² who has examined fragments of mucosa found accidentally in the wash water, there is in more than one-half the cases of hyperacidity "a proliferation of the glandular elements and increase of oxyntic or border cells." Straus³ also describes similar lesions.

Symptoms.—The subjunctive symptoms usually appear gradually; they consist of acid eructations, heartburn, and pain and burning in the stomach. Acid eructations usually appear at the height of the paroxysms of gastralgia, and their appearance is accompanied by a passing relief from pain. If portions of the acid secretion of the stomach are eructated, the mucous membrane of the oesophagus is subjected to this irritation, and heartburn develops. This symptom usually appears after eating acid foods, and is frequently relieved by taking milk, meats, and eggs. The pain may vary from a severe pressure in the stomach to a very acute pain, and may extend into the back between the shoulder-blades and pass under the sternum to the pharynx (pyrosis hydrochlorica). It is not continuous, but appears at certain periods with intervals of complete relief, usually from two to four hours after meals, depending upon the quality and quantity of food taken. In a very small number of cases the pain supervenes immediately upon eating, as occurred in 9 of the series (1.6 per cent.). The symptoms continue for a variable period of time, disappearing in the course of an hour or persisting for many hours. Pain is much more easily induced by starchy foods than by protein foods, more quickly by light meals than by heavy ones, and is usually relieved by the ingestion of food or by neutralizing the acid by means of some alkali. Paroxysms of pain (gastralgia) occur in a certain proportion of cases; they develop gradually, beginning as a feeling of pressure and becoming more severe. As a rule, the pain disappears at night upon lying down, and does not begin again until several hours after breakfast.

The severe gastralgic paroxysms are not commonly found after every meal. They are accompanied not only by intense pains in the epigastrium, but also in the back; they are partially relieved by the acid eructations and entirely by the vomiting of the acid secretion, which burns the throat and

¹ *Deut. med. Wochenschrift*, 1895, Nr. 21.

² *Archiv f. Verdauungsk.*, Bd. iv, S. 23; *Diseases of the Stomach*, third edition, p. 814.

³ *Virchow's Archiv*, 1898, Bd. cliv.

numbs the teeth as it passes over them. In some cases the seizures may be very mild, and in other cases they recur irregularly at intervals of days. In this series of 542 cases, gastralgic seizures occurred in 176, *i. e.*, 32 per cent. A sensation of burning in the stomach is frequently observed; this is felt in the epigastrium, and may extend to the back. It is relieved by the same measures as those which relieve the pain, namely, by alkalies or the taking of some form of food. In this series there were 221 that complained of this sensation, *i. e.*, 40 per cent. The appetite is usually good, and as eating usually relieves the symptoms, the patient often accustoms himself to eat frequently; small quantities will, however, often satisfy the patient. In 124 (21 per cent.) of the cases the appetite was increased, in 357 (66 per cent.) it was normal, and in 61 (13 per cent.) it was diminished. The thirst is not greatly increased in this disorder, although the reverse is believed to be the case by most authorities. In this series thirst was only increased in 31, *i. e.*, 6 per cent.

The bowels are frequently constipated, as occurred in 164 of the series, or 30 per cent. We have already pointed out the etiological relationship of this condition to hyperchlorhydria. Occasionally constipation alternates with diarrhoea, as occurred in 7 of the cases (1.3 per cent.).

The objective symptoms may be divided into two classes, those presented by the physical examination of the patient and those obtained by the examination of the vomited material and gastric contents. The patient, as a rule, is in good general health, has lost but little flesh, and the general nutrition is not usually disturbed; the abdomen is not usually sensitive to pressure, and painful areas are not apparent except perhaps during the attacks of pain, when the stomach may be found distended and sensitive to pressure. In some patients an atony of the stomach may be detected which may bear some etiological relation to the hyperchlorhydria. Vomiting is not frequent; it usually occurs when the paroxysms of gastralgia are present, and then at the height of the attack; relief is obtained by vomiting, and the pain disappears. This symptom occurred in 86 of the cases, or 15 per cent. The vomited matter is very acid, which is detected even by the patient himself; on examination this material is found to contain large quantities of free hydrochloric acid. The examination of the gastric contents after a test meal usually reveals a normal or hypermotility. Six hours after a Riegel test dinner the stomach is usually empty; one hour after a test breakfast it contains only a small quantity of gastric juice. In 30 per cent. of this series there was an atony of the stomach in which more or less retention was observed. The gastric juice shows a very high degree of acidity; the starch digestion is imperfect, and rather large quantities of amidulin are found, as the excess of acid impedes the action of the ptyalin. On the other hand, the proteid digestion in the stomach is complete and peptonization is more rapid than under normal conditions, so that none or but few undigested meat fibers are found in the gastric contents three to four hours after a test dinner, while amylaceous food is present unchanged and in abundance.

Schüler¹ has pointed out that the specific gravity of the gastric juice is diminished in hyperchlorhydria. According to this observer it is usually below 1016, the normal specific gravity varying between 1016 to 1020. The observations in 14 cases as to the specific gravity of the gastric contents

¹ *Deut. med. Wochenschrift*, May 10, 1900.

in this condition are, on the whole, in accord with those of Schüler, ranging between 1008 and 1014, although occasionally higher figures are found, as in 3 of this series between 1019 and 1022.

Absorption in the stomach is said to be more rapid in hyperchlorhydria than under normal conditions.

The urine in hyperchlorhydria is at times alkaline and contains a precipitate of phosphate, especially after severe attacks of vomiting. The acidity of the urine is diminished proportionately to the increase in acidity of the gastric juice, and the chlorides are also diminished in quantity.

Diagnosis.—This is established by careful investigation into the subjective symptoms of the patient together with the results obtained from an analysis of the gastric contents. These symptoms consist of acid eructations, heartburn, burning and pains in the stomach, the pain appearing from two to four hours after meals, relief being obtained by the ingestion of food and by neutralizing the acid with alkalis.

The examination of the stomach contents after a test meal reveals an increase in free hydrochloric acid; while on fasting, the stomach is found nearly or almost completely empty. Hyperchlorhydria must be differentiated from chronic hypersecretion, ulcer, acid gastritis, atony, and cholelithiasis.

Chronic hypersecretion is distinguished by the intense gastric pains appearing frequently at night, the more frequent vomiting, and the appearance of large quantities of gastric juice (100 cc. and more) in the fasting stomach. *Ulcer* of the stomach (without hemorrhage) is differentiated by the presence of the circumscribed epigastric and dorsal areas of pain. The pain in ulcer is proportionate to the quality and quantity of the food taken, which is not usual in hyperchlorhydria; while the pain of hyperacidity is relieved by alkalis, which is not the case in ulcer. Occult blood is frequently found in the feces in ulcer, but not in hyperchlorhydria. The differential diagnosis between these two conditions is very difficult at times, as hyperacidity frequently accompanies ulcer of the stomach; the greater irregularity of the symptoms in hyperchlorhydria and presence at least of some of the definite symptoms of ulcer will usually lead to a correct diagnosis. In doubtful cases, Boas advises that the patient undergo the rest treatment for ulcer; a favorable outcome would rather indicate the presence of ulcer.

Hyperchlorhydria is distinguished from an *acid gastritis* by the constant absence of mucus in the gastric contents, and from *atony* by the absence of symptoms of retention. In a certain proportion of cases of hyperchlorhydria, however, atony is present (30 per cent.) and the symptoms of both conditions become marked. In *cholelithiasis* the pains are not relieved by the ingestion of food or the administration of alkalis, and radiate to the right toward the hypochondriac region and are in no way dependent upon the time of the ingestion of food, whereas the pains of hyperchlorhydria usually appear at the height of digestion, *i. e.*, from two to four hours after meals.

Prognosis.—This is usually quite favorable as to the relief of the symptoms, especially if the condition is of recent origin; in protracted cases, however, the prognosis is less favorable, and even when patients have been apparently cured of this condition relapses are not infrequent, probably due in many instances to the fact that while at first the acidity of the gastric secretion may be reduced, a permanent reduction is not usually effected.

Riegel,¹ Kauffmann,² and Steele³ believe that it is impossible to permanently reduce the secretion of acid, but do not produce any observations made in this direction. In personal observations in 9 patients in whom we have been able to make gastric analyses more or less frequently during the time of treatment, *i. e.*, for a period of six months, and again after the first and second years, during which symptoms of hyperacidity no longer existed and treatment was no longer instituted, it is evident that while the patient is under treatment and the acidity of the gastric juice can be reduced, this reduction is not permanent, and that while the patient may be free of all symptoms of hyperacidity an excess of acid secretion still exists. This points to the fact that our treatment is only effectual in reducing the gastric hyperæsthesia and not the acidity. The following table represents the observations in the 9 cases to which reference has been made:

Case.	Original per cent. of free HCl.	Average per cent. of free HCl during first 6 months of treatment.	Per cent. of free HCl after end of first year, all treatment having been suspended; no symptoms.	Per cent. of free HCl about end of second year, all treatment having been suspended; no symptoms.
1	0.28	0.15	0.24	0.23
2	0.25	0.19	0.27	0.28
3	0.24	0.16	0.23	0.21
4	0.27	0.17	0.29	0.27
5	0.31	0.18	0.32	0.23
6	0.30	0.28	0.29	0.26
7	0.26	0.20	0.25	0.26
8	0.27	0.18	0.29	0.25
9	0.25	0.17	0.27	0.29

In those instances in which the hyperacidity is symptomatic of other conditions the prognosis depends upon the relief of the primary disease. In the series of 542 cases of hyperchlorhydria in which it was possible to follow 48 cases for a period of at least three years or more, 31 were cured (65 per cent.), 10 were relieved (21 per cent.), and 7 were not benefited (14 per cent.); relapses occurred in 5 cases (10 per cent.).

Treatment.—In this the diet plays the most important role, it being essential that the food should be given in such a form as not to produce any irritation to the mucous membrane of the stomach. As the gastric hyperæsthesia is mainly responsible for the hyperacidity, the treatment should be directed to this condition. This would include the removal of all causes of irritation, the prohibition of alcoholic stimulants to a large degree, these to be taken only occasionally and in small quantities; all acids, including organic acids, should be forbidden; all spices and condiments, such as ginger, pepper, mustard, and horseradish, should be prohibited. All foods to which vinegar or lemon juice have been added should be interdicted; all hard substances which are apt to irritate the stomach, such as nuts, should be avoided; food must be thoroughly masticated and should be taken neither too hot nor too cold. Formerly a diet consisting almost exclusively of proteids was recommended in order to neutralize as much acid as possible, as the proteids possess the power of combining with free hydrochloric acid. On the other

¹ *Deut. med. Wochenschrift*, May 12, 1904.

² *Zeitschrift f. klin. Med.*, vol. lvii, p. 491.

³ *Journal of the American Medical Association*, August 18, 1906.

hand, it was soon discovered that while the proteids possess this power, they nevertheless again stimulate the flow of acid, some, such as raw beef, beef-juice, and beef extract, more than others, such as well-cooked meat.

More recently various authors, such as v. Schlern, Jurgensen, Hemmeter, Backmann, and others recommended a carbohydrate diet in preference to a proteid one. There can be no question that carbohydrates are far better borne in superacidity than was formerly supposed. Recent writers, such as Boas and Steele, prefer a liberal mixed diet, consisting of proteids, fats, and carbohydrates. As regards the proteid food, Fleischer¹ has investigated the subject of the combining effect of muriatic acid with various foods. He discovered that beef, veal, ham, and mutton bind twice as much muriatic acid as do calf's brain and sweetbreads. The following table taken from Fleischer shows the ability of various foods to combine with muriatic acid:

Meats (100 grams).	Pure HCl.	25 per cent. HCl.	Dilute muriatic acid.
Calf's brains, boiled	0.65	2.60	5.20
Liver sausage	0.80	3.20	6.40
Calf's thymus, boiled	0.90	3.60	7.20
Meat sausage	1.00	4.00	8.00
Blood sausage	1.30	5.20	10.40
Pork, boiled	1.60	6.40	12.80
Ham, boiled	1.80	7.20	14.40
Ham, raw	1.90	7.60	15.20
Mutton, boiled	1.90	7.60	15.20
Beef, boiled	2.00	8.00	16.00
Veal, boiled	2.20	8.80	17.60
Leube-Rosenthal meat solution	2.20	8.80	17.60
Beer	0.10	0.40	0.80
Milk	0.36	1.20	2.40
White bread	0.30	1.20	2.40
Graham bread	0.30	1.20	2.48
Black bread (gray bread)	0.50	2.00	4.00
Pumpernickel	0.70	2.80	5.60
"Hand" cheese	1.00	4.00	8.00
Fromage de Brie	1.30	5.20	10.40
Edam cheese	1.40	5.60	11.20
Swiss cheese	2.60	10.40	20.80
Cocoa L	4.10	16.40	32.80

The carbohydrate foods which are allowable in this condition are the more digestible vegetables, such as mashed potatoes, spinach, asparagus, peas, and carrots, strained and eaten in the form of purees, also the digestible farinaceous foods, such as rice and grits. Strauss² has recommended the administration of a solution of sugar in this condition; this food can be best given in most instances in the form of some baked cake in which part of the starch has become dextrinized by baking or roasting. Strauss and Aldor have advised the use of fats in hyperchlorhydria, inasmuch as they tend to lessen the acidity of the gastric secretion. They are best given in the form of butter, cream, olive oil, and the like. Of the fluids, milk, cocoa, and alkaline mineral waters, such as Apollinaris, Vichy, and Seltzer, are especially useful; the carbon dioxide contained in these waters produces a sedative effect and lessens the secretion of acid. These waters may be used to dilute wine and milk.

¹ *Krankheiten d. Speiseröhr. d. Magens u. d. Darms*, 1896, p. 932.

² *Zeitschrift f. klin. Med.*, vol. xxix.

In arranging the diet for patients with hyperchlorhydria it has been found best in dealing with patients taking but little nourishment to allow them to eat at frequent intervals; if, however, large meals are consumed, it is advisable to permit only three meals a day, allowing the stomach to rest in the intervals. In advanced cases, with marked emaciation and prominent nervous symptoms, an absolute or modified rest cure will accomplish excellent results; in these cases milk and egg albumin should be the only food given at first, gradually increasing, and finally allowing easily digestible solid food.

The following list, taken from Friedenwald and Ruhräh,¹ gives the foods which are allowable and those which are forbidden in hyperchlorhydria: Allowable: Eggs, soft boiled, hard boiled, or poached. Meats, boiled or broiled; brains, raw scraped beef, boiled or broiled beef, broiled steak, roast mutton, broiled chops, roast lamb; boiled, broiled, or roasted chicken; broiled or roasted squab, roast turkey, broiled or roasted birds. Farina-cious food: Rice, cornstarch, sago, tapioca, arrowroot, hominy, grits, vermicelli, cream of wheat, stale wheat bread, toast corn bread, pulled bread, zwiebach. Fruits: Baked or stewed apples, stewed apricots, stewed peaches, stewed pears, stewed prunes. Fatty foods: Butter, cream, pure olive oil. Drinks (taken mainly between meals): Milk, buttermilk, malted milk, peptonized milk, milk with lime-water, milk with Vichy, milk flavored with tea, milk flavored with coffee, kefir, koumyss, junket, whey, cocoa, albumin-water, water (not with meals), hot water. Mineral waters: Vichy, Apollinaris, Poland, Lithia water, Congress Hathorne, Carlsbad.

They must not take: Soups, fried foods, pork, veal, stews, hashes, corned meat, liver, kidney, duck, goose, sausage, crabs, lobsters, preserved fish, smoked fish, salmon, salt mackerel, sardines, cauliflower, celery, cocoa, radishes, cucumbers, sweet potatoes, beets, tomatoes, acid fruits, salads, hot bread or cakes, nuts, candies, pies, pastry, strong tea, coffee, alcoholic stimulants, ice-water, ice-cream.

Hygienic Measures.—General hygienic measures should never be neglected in the treatment of hyperchlorhydria. Those patients who have been fatigued by overwork should be ordered rest, and those who are worn out by mental strain and excitement are often best ordered away to the country, seashore, or mountains; physical exercises, outdoor life, and cold sponge baths are often serviceable adjuvants; warm poultices or Preissnitz compresses are frequently useful in the relief of pain.

Gastric lavage is rarely necessary in the simple forms of hyperchlorhydria, although it is often of benefit in severe and obstinate forms. It is best practised late in the evening three or four hours after the last meal, or on an empty stomach in the morning; occasionally douching of the stomach with a solution of silver nitrate may be beneficial; 500 cc. of a 1 to 2000 solution is allowed to enter the stomach and again washed out thoroughly with normal salt solution.

Electricity often exerts a very beneficial effect in some cases of hyperchlorhydria, and can be used either as the intragastric faradization or galvanization. The faradic current is useful in most instances, but in painful conditions the galvanic current is to be preferred. The chronic constipation often associated with hyperchlorhydria is frequently relieved by this mode of treatment.

¹ *Diet in Health and Disease*, second edition, p. 688.

Medicinal Treatment.—Alkalies are employed to neutralize the excess of acid in hyperchlorhydria. Of these, the bicarbonate of soda, calcined magnesia, magnesia carbonate, calcium carbonate, and the phosphate of soda are most frequently utilized. Calcined magnesia has an advantage over bicarbonate of soda, as the former neutralizes four times as much acid as the latter; it has the additional advantage in that it is distinctly laxative and is therefore especially useful when constipation is present; the doses of the alkali should be, so far as possible, proportioned to the heaviness of the meal as well as the degree of hyperacidity. The alkalis should be administered when the discomfort begins to manifest itself, *i. e.*, about two hours after meals. Of other drugs found useful in the conditions are:

1. Atropine, which inhibits gastric secretion. This remedy can be administered in the form of the extract of belladonna or atropine.

2. Nitrate of silver in solution, which tends to diminish the gastric hyperæsthesia.

3. Bismuth subnitrate and carbonate, which may be administered in large doses and which exert a beneficial, largely mechanical, effect on the gastric mucosa.

4. The nerve sedatives, such as the bromides, valerianates, and sumbul, which relieve the gastric hyperæsthesia due to nervous irritation. In those cases in which symptoms of hyperacidity exist and in which the gastric secretion shows a normal or lessened amount of acid the sedatives are more valuable than the alkalies.

5. Nux Vomica. While the bitter tonics are to be avoided, as a rule, in hyperchlorhydria, Musser¹ advises the administration of nux vomica in ascending doses in those cases in which the symptoms of hyperacidity are due to a sensory neurosis; he has received uniformly good results from this form of treatment.

6. Olive Oil. Cohnheim² has recommended olive oil very highly in the treatment of this condition. It can be administered through the stomach tube, 100 to 200 cc. being introduced each morning, or it may be given in tablespoonful doses before meals or about two hours after meals. The oil coats over the mucous membrane and lessens the acid secretion.

Inasmuch as constipation is sometimes the cause of hyperchlorhydria, a correction of this will often give permanent relief from the hyperacidity, and is often best accomplished by diet rather than by drugs. In a not inconsiderable number of cases, as has been pointed out by Epstein,³ very excellent results are obtained by means of the large oil enemata of Fleiner, not only as regards the relief of this constipation, but also of the hyperacidity. Meunier⁴ has shown that chewing gum or some similar substance after meals has a marked influence on the digestion of starch, promoting it by the large supplementary amounts of saliva swallowed, thus largely overcoming the excess of acid in hyperchlorhydria.

Of the watering places for the treatment of hyperchlorhydria, Karlsbad, Vichy, Neuenahr, and Bedford and Saratoga (Vichy) are to be most highly recommended. The use of these waters when taken judiciously has a ten-

¹ *Transactions of the Association of American Physicians*, 1905, p. 193.

² *Zeitschrift f. klin. Med.*, vol. iii, S. 110.

³ *Deut. med. Wochenschrift*, 1904, S. 1749.

⁴ *Presse Médicale*, 1906, No. 102.

dency to relieve the symptoms in hyperacidity; they act not only by reducing the degree of acid of the gastric secretion, but also by subduing the gastric hyperæsthesia; the laxative effect of some of these waters is also useful in the treatment of many of these cases.

GASTROSUCCORRHŒA OR HYPERSECRETION OF GASTRIC JUICE.

Gastrosuccorrhœa or hypersecretion of gastric juice is a condition in which a constant excessive flow of gastric juice is secreted. The stomach pours out this secretion even when free from food, so that large quantities of gastric juice may be found even in the morning before the ingestion of any nourishment. This disease was first described by Reichmann¹ in 1882, and afterward more fully elucidated by Riegel in various communications between 1884 and 1893, as well as by Honigsmann,² Sticker,³ Jaworski,⁴ Van den Velden,⁵ and Bonveret and Devic.⁶

The disease is found in three forms: gastrosuccorrhœa continua periodica, gastrosuccorrhœa continua chronica, and digestive gastrosuccorrhœa. There has been considerable diversity of opinion among various authors whether gastrosuccorrhœa should be placed in the category of organic diseases or among the neuroses. There is no question that in many instances this condition is a result of some marked organic gastric affection, as ulcer with obstruction, dilatation, or atony. On the other hand, there are a certain number of cases in which the condition can only be looked upon as neurotic; this is more frequently the case with the form known as gastrosuccorrhœa continua periodica than the continua chronica.

Gastrosuccorrhœa Continua Periodica.—By this is meant that condition in which, in addition to the appearance of the acute attacks with a constant secretion of gastric juice, severe gastric pains and vomiting are present. Rossbach⁷ has called attention to a condition which is closely related to periodical hypersecretion, but in which the attack takes its onset with violent headaches and which he terms gastroxynsis. Periodical hypersecretion may be present as a simple gastric neurosis or as a reflex neurosis, secondary to disease of the brain or spinal cord, *i. e.*, progressive paralysis, tabes dorsalis, or myelitis.

Etiology.—The causes of intermittent hypersecretion are various, among which may be mentioned excessive mental strain, excitement, anger, over-indulgence in food, and the abuse of tobacco. At times this condition is found in connection with the crises of locomotor ataxia, accounted for by the fact that the nerve fibers leading to the gastric glands regulating the flow of hydrochloric acid are unduly stimulated.

The patient suffering with this condition usually presents other nervous symptoms indicating the nervous origin of the disorder. The disease occurs usually in young people, and more frequently in males than in females. In

¹ *Berl. klin. Wochenschrift*, 1882, Nr. 40.

² *Münch. med. Wochenschrift*, 1887, Nr. 48, etc.

³ *Ibid.*, 1886, Nrs. 32 und 33.

⁴ *Zeitschrift f. klin. Med.*, Band ii, Hefte 2 und 3.

⁵ *Volkmann's Sammlung klin. Vorträge*.

⁶ *La dyspepsie par hypersecretion gastrique*, Paris, 1892 (monograph).

⁷ *Deut. Archiv f. klin. Medicin*, 1885, Band cclvii.

a series of 1592 cases, 21 suffered with intermittent hypersecretion (1.5 per cent.). The following table represents the number in males and females at various ages:

Age.	Males.	Females.
20 to 30	7	3
30 to 40	5	3
40 to 50	2	0
50 to 60	1	0

Symptoms.—The characteristic signs of this condition are not only the paroxysmal appearance of the attacks, which take their onset in the midst of perfect health, but also the special character of the gastric contents and vomitus. The attack has its onset early in the morning, usually with lassitude, malaise, headache, loss of appetite, thirst, and pains in the stomach, which become intense and are accompanied by heartburn and acid belching. The pains become spasmodic and more intense, and finally vomiting sets in. The vomited matter is very acid, at first consisting of food and then of gastric juice. The attacks may thus be entirely recovered from, or after a short period of relief others may set in, extending over a period of from a few hours to several days. The vomiting can be prevented at times by the drinking of large draughts of water or by the administration of large doses of alkalis. The quantity of the vomited matter is usually very large in amount, often from 200 to 500 cc., containing at first food particles and afterward consisting of pure gastric juice, which is tinged yellowish or greenish with bile. The material first vomited is usually very acid, often containing 3 per cent. or more free hydrochloric acid, but afterward, when admixed with bile, the acidity is greatly reduced to or below normal. The vomited material may contain slight traces of blood, the appearance of which has no special significance. During the attack, besides the intense pain there is a total loss of appetite, great thirst, and general feebleness; the pulse becomes weak and the patient presents the appearance of great suffering; he becomes pale and breaks out in a cold perspiration. The urine is greatly diminished in quantity and of a high specific gravity, while the bowels are constipated. The attacks vary greatly both as to intensity and duration, sometimes terminating gradually, sometimes suddenly. Between the attacks the patient is usually in good health, free of all disturbances, although at times moderate gastric disturbances exist, such as discomfort in the stomach, acid eructations and heartburn. An analysis of the gastric contents at this time usually reveals a hyperacidity, even though symptoms of hyperchlorhydria are not present. The condition known as gastroxynsis differs from periodic hypersecretion only in that the headache is a most important and persistent symptom. Rossbach found this condition in nervous individuals who had overexerted themselves. The other symptoms of gastroxynsis are so similar to periodic hypersecretion that these cases should be classed in the same category. The following represents a typical instance of periodic hypersecretion:

J. S., a lawyer, aged forty-one years, has always been in good health, with the exception of attacks which have been coming on at irregular intervals for the past three years, usually appearing after some great mental strain. At first they appeared at intervals of from five to six months, but recently have been much more frequent, coming on every two or three weeks. The

onset is characterized by indigestion, pressure, fulness in the abdomen, nausea, and heartburn; finally vomiting occurs. The vomited matter consists of a large quantity of very acid gastric juice; the attack is accompanied by very severe abdominal pain. The patient is greatly prostrated by the attack and is forced to take to his bed, and hypodermic injections of morphine must be administered in order to give even temporary relief. The attacks last from three to seven days. On examination two days after the onset of the attack, the patient was found greatly prostrated, with a rapid (118 per minute) and feeble pulse, and suffering from severe depression. The heart and lungs were normal. The abdomen was everywhere sensitive to pressure; the stomach was not found enlarged; without the ingestion of food 150 cc. of gastric secretion were obtained by means of the tube, with a total acidity of 90; free hydrochloric acid, 0.26 per cent.; the vomited material presents a total acidity of 50; free hydrochloric acid, 0.12 per cent. Several examinations in other attacks revealed similar conditions. The patient was kept under the influence of opiates, and recovered in three days. The examination of the gastric contents one hour after an Ewald test breakfast, three days after the disappearance of the attack, showed a total acidity of 84 and free hydrochloric acid 0.25 per cent. After a sea voyage of one month and with carefully regulated habits, as to diet, etc., and the use of intragastric faradization, the attacks became far less severe and less frequent.

Diagnosis.—The diagnosis of periodic hypersecretion is made by the appearance of the symptom-complex already described, as well as by the finding of large quantities of gastric juice in the stomach at a time when no ingesta are present. The differential diagnosis must be made from periodic vomiting and the vomiting occurring in the gastric crises of locomotor ataxia.

Periodic hypersecretion is distinguished from periodic vomiting by the irregular appearance of the attacks and by the vomiting of large quantities of pure gastric juice, whereas, in periodic vomiting, the attacks appear at regular intervals and the vomited material consists of mucus and bile. From the vomiting occurring in the gastric crises periodic hypersecretion is also differentiated by the appearance of large quantities of acid gastric secretion in the vomit and by the fact that the attacks persist during the night, neither of which is usual in the crises of locomotor ataxia.

Prognosis.—The duration of the disease varies greatly; some attacks are short, lasting but a few hours, others extend over a period of days. Sometimes the patient becomes greatly weakened and prostrated by the effect of the attack; in other instances his general health will in no way be impaired. If the primary cause of the disease can be determined and relieved the prognosis is favorable; on the other hand, this is impossible in many instances, and notwithstanding all treatment, the attacks reappear at intervals of weeks, months, or even a year.

Treatment.—The treatment of periodic hypersecretion is, so far as possible, to seek out and relieve the cause. If the condition is due to overanxiety or mental strain, the patient should be ordered to the mountains or seashore. Physical exercises should be insisted on, and overindulgence in food and drink and in the use of tobacco should be prohibited. Hydrotherapy exerts a markedly favorable influence in the relief of this condition.

In a number of our cases relief was afforded by means of intragastric electricity. In those instances in which the attacks have their origin from

a hyperchlorhydria this condition must be treated. For the attack itself the mode of treatment advised by Einhorn¹ is often found serviceable; that is, to administer a moderate dose of bromide just as the attack begins; this method will at times abort an attack. As soon as the attack sets in the stomach should be thoroughly washed out with an alkaline solution, and this may be repeated a number of times during the attack. For the severe pain one is often forced to administer morphine hypodermically, it being the only remedy that can be relied upon at times to produce even temporary relief. During the attack the patient should be allowed but small quantities of fluids, water, milk, and egg albumin; bits of ice may be administered to relieve the thirst; also enemata of normal salt solution may be given.

Gastrosuccorrhœa Continua Chronica.—This is also known as Reichmann's disease, or chronic hypersecretion, and also as chronic continuous secretion of gastric juice. It is characterized by a chronic continuous secretion of gastric juice, even in the absence of food in the stomach, that is in the fasting stomach. This condition may be primary or secondary. It is primary when it occurs as a gastric neurosis independent of any known anatomical change in the stomach; it is secondary when it occurs as the result of some other gastric disturbance, such as ulcer of the stomach, dilatation, or atony. As continuous hypersecretion is most frequently associated with dilatation, and as the symptoms of both conditions are very similar, a number of authors deny the existence of this condition as a separate entity and believe that it is simply a form of dilatation. Thus Schreiber and Martius altogether deny the existence of this condition, believing it to be identical with gastric dilatation, while Riegel and Reichmann claim the contrary. It cannot be denied that most cases of chronic gastrosuccorrhœa are secondary to dilatation, but nevertheless, there are a certain number which are in no way associated with dilatation or even atony of the stomach, and which are primary in character and of a neurotic type. Boas² has also recently come to this conclusion.

The difficulty in differentiating between continuous hypersecretion and dilatation is more evident when we observe that of the six cases of this disease originally reported by Reichmann,³ but one is a case of chronic hypersecretion, the remainder presenting symptoms of hypersecretion secondary to pyloric stenosis. Riegel⁴ and Einhorn⁵ believe that continuous hypersecretion is in some instances a perversion of function and really of a neurotic type, in which no organic lesion can be detected. M. Pickardt⁶ has also recently reported three cases in which this condition was primary and existed as a pure neurosis, and in which careful investigation failed to show any organic disease of the stomach or motor insufficiency. The cases are further interesting inasmuch as the patients all belong to one family, being a mother and two sons.

Etiology.—The causes of continuous hypersecretion are much like those of intermittent gastrosuccorrhœa, namely, great mental anxiety and excitement, indiscretion in the use of food, and the abuse of drink, extending over

¹ *Diseases of the Stomach*, fourth edition, p. 362.

² *Deut. med. Wochenschrift*, January 27, 1907, S. 127.

³ *Berliner klin. Wochenschrift*, 1882, Nr. 40; 1884, Nr. 48; 1887, Nr. 12.

⁴ *Diseases of the Stomach*, p. 334.

⁵ *New York Medical Record*, January 13, 1906.

⁶ *Berliner klin. Wochenschrift*, October 30, 1905.

long periods of time. Nervous symptoms are usually present, indicating the nervous origin of the disorder. Secondary continuous hypersecretion may arise as a result of dilatation, atony, or ulcer. Primary continuous hypersecretion is a rare disease; it does not occur as frequently as intermittent hypersecretion; it is found more frequently in males than in females, and more often in the young and middle-aged. In this series of 1592 cases there were 10 cases of primary continuous hypersecretion (0.65 per cent.), 7 in males and 3 in females. Four were between twenty and thirty years of age, 3 between thirty and forty, and 3 between forty and fifty.

Pathology.—The anatomical changes in these conditions are not always uniform. Korezynski and Jaworski,¹ who have investigated the subject most carefully, find no anatomical changes whatever in a certain number of cases. In others a special form of gastritis existed, associated with a degeneration and destruction of the peptic cells, while the parietal cells remained normal. Interstitial changes were also observed. Hayem,² Cohnheim,³ and Hemmeter⁴ have observed similar changes.

Symptoms.—The subjective symptoms of chronic hypersecretion are pain, burning in the epigastrium, heartburn, acid eructations, nausea, vomiting of large quantities of gastric juice, and increased thirst. These make their appearance gradually, and many patients complain of mild dyspeptic symptoms for years before the true nature of the disease is revealed. At first there is but a slight degree of burning in the epigastrium and heartburn, which gradually intensifies, then pain appears, coming on several hours after meals, and relieved by the ingestion of food. The pain also appears in attacks during the night, a rather characteristic symptom of this disease. After the pain has existed for some time, vomiting sets in, the vomitus consisting of a large quantity of a very acid gastric juice.

These symptoms may be mild in some patients and very severe in others; they are apt to appear for a certain length of time and disappear, to recur after weeks or months; in severe attacks the symptoms are present almost daily, and vomiting may occur four or five times a day. The patient loses flesh, the tongue and skin become dry, the appetite is increased in some cases, diminished in others. The attacks of pain can be often lessened by the ingestion of proteid food, such as boiled eggs or milk. Constipation is usually present, and the quantity of urine is diminished, the chlorides decreased, and the phosphates increased. The subjective signs consist in demonstrating hyperacidity as well as the presence of large amounts of gastric juice in the fasting stomach. The acidity of the gastric juice after a test breakfast may amount to 80, 90, 100, or more, and the free hydrochloric acid to 0.25 to 0.3 per cent. The proteid digestion is good, showing a perfect peptone reaction, while the starch digestion is imperfect, the continuous flow of acid impeding amylolysis. The most important and characteristic symptom is the finding of gastric secretion in a fasting stomach; under normal conditions small quantities of gastric juice may be found in the stomach, but when quantities of 100 cc. or more are present the condition of hypersecretion must be considered, and especially so if it is discovered at a number of examinations. It cannot be denied, however, that even when smaller quantities than 100 cc. are discovered and when the other

¹ *Deut. Archiv f. klin. Med.*, vol. xlvii.

³ *Archiv f. Verdauungskr.*, 1896, Bd. i.

² *Gaz. hebdom.*, 1892, Nr. 33 to 34.

⁴ *Ibid.*, Bd. iv.

symptoms of continuous hypersecretion are present, these cases also must be classified under the head of chronic continuous gastrosuccorrhœa.

In order to arrive at a more certain diagnosis, lavage should be given the evening before; the next morning the stomach is tested, fasting, so that no portions of former meals may be retained. The gastric secretion obtained from the fasting stomach is usually watery and often contains a small trace of bile; it is of a high total acidity and contains an excess of free hydrochloric acid, the peptone reaction being good, the dextrin reaction absent. In the secondary form of gastrosuccorrhœa continua chronica, dilatation or atony of the stomach is often present. In these cases in addition to the usual signs of the disease, marked evidences of these special conditions are present.

Sequelæ.—Of the sequelæ or complications, atony and dilatation of the stomach, with or without ulcer, are the most common. Atony and dilatation often appear after the gastrosuccorrhœa has existed for a considerable period of time; it is only in the earlier stages that the primary nature of the chronic hypersecretion can be determined; when the patient is seen late in the course of the disease, when both conditions are marked, it is often impossible to determine which condition is primary and which is secondary.

Diagnosis.—This is suggested by the symptoms, *i. e.*, excessive pain occurring several hours after meals and also during the night, with vomiting of large quantities of gastric juice, which shows an excellent digestion of proteids, but a deficient starch digestion. In addition there is burning in the epigastrium, heartburn, acid eructations, and increased thirst. The diagnosis is made certain by examining the stomach in the fasting state, when large quantities of gastric juice, usually over 100 cc., are obtained, showing an entire absence of amylolysis. It is important in these cases to practice lavage on the evening before the morning that the fasting stomach is tested. In regard to the differential diagnosis it is often important to exclude atony and dilatation, which, however, are often associated with gastrosuccorrhœa. In atony a motor insufficiency is observed, while in dilatation the motor insufficiency is so great that food particles are still present in the fasting stomach. This is not the case in gastrosuccorrhœa unless the condition is accompanied by dilatation. Ulcer will be excluded by the absence of hemorrhage, a circumscribed painful area, and the presence of large quantities of gastric juice in the fasting stomach.

Prognosis.—This depends largely upon whether the condition is primary or secondary. If it is secondary to a dilatation, the prognosis is not as favorable as if it were primary, unless some radical procedure is undertaken, such as a pyloroplasty or gastro-enterostomy. When primary, the prognosis is usually not unfavorable, especially if rational treatment is undertaken. Frequent relapses, however, are common, while certain severe cases are often very protracted and may continue indefinitely, notwithstanding the most rigid form of treatment.

Treatment.—This consists in relieving the irritation of the gastric mucosa and thus diminishing the flow of gastric secretion. Diet is of primary importance in this regard. This should consist of small but frequent meals given at intervals of from three to four hours. The patient should be required to eat slowly and masticate his food thoroughly; all irritating substances such as pepper, mustard, onions, and highly seasoned foods should be interdicted. As motor disturbances are apt to occur in this condition, fluids should be administered sparingly. The diet should be made up mainly

of proteins, since these foods are well borne. The carbohydrates are poorly digested, and hence must be given only in small quantities and in the most easily digestible forms. All forms of meat are allowable, roast beef, lamb chops, chicken, and broiled steak, but should be finely minced. Of the carbohydrates the best are oatmeal, zwieback, Nestle's food, purée of potatoes, spinach, etc., may also be allowed. Fats may be permitted to a greater or less degree in the form of good butter, cream, or olive oil. Milk is an excellent form of food in this disease, and may be taken either alone or mixed with small quantities of tea, coffee, or cocoa. It may also be given with eggs.

The foregoing regulations as to diet apply to chronic hypersecretion without dilatation; when cetasia exists the dietetic rules described under this head must be followed.

Lavage of the stomach is a most efficient means of relieving pain and irritation. This is best practised in the morning before the ingestion of food. Instead of lavage Boas recommends emptying the stomach in the fasting state (expression) by means of the tube; while Reichmann believes in lavage of the stomach with solutions of nitrate of silver 1 or 2 to 1000. Penzoldt advises douching with a solution of boric acid, and Einhorn sprays the stomach with nitrate of silver.

The alkalies play a most important role in the treatment of the disease; of these, bicarbonate of soda, calcined magnesia, phosphate and citrate of soda are most commonly employed. These are best administered two to three hours after meals, *i. e.*, at the height of digestion. Other remedies which are useful at times in this condition are bismuth subnitrate given in large doses (half a dram), belladonna, eumydrin, and nitrate of silver in solution.

Einhorn recommends the use of direct galvanization of the stomach, from which gratifying results may be obtained in these conditions.

Digestive Gastrosuccorrhœa.—Straus¹ first pointed out that in certain cases of hyperacidity relatively large quantities of a thin, watery, gastric secretion were obtained one hour after a test breakfast. He called this condition digestive or alimentary hypersecretion. Zweig and Calvo² working under the direction of Boas, investigated this subject more fully, and discovered that the large amounts of contents obtained after the test breakfast do not depend upon motor disturbances of the stomach, such as atony, but rather upon increased gastric secretion. Elsner³ has also called attention to this condition. Boas⁴ has recently investigated this question, and considers the name digestive gastrosuccorrhœa more appropriate than alimentary gastrosuccorrhœa, because the increased gastric secretion is dependent upon the act of digestion in contradistinction to continuous hypersecretion. In the latter the secretion extends beyond the digestive act, and more or less gastric juice is found in the fasting stomach.

Etiology.—This disease occurs most frequently in males. The 12 cases of Boas all occurred in males, and both of the patients in this series were males. Age plays no important role, according to Boas; the disease is found in youth as well as in old age. In this series the ages were twenty-eight and forty-four years respectively.

¹ *Deut. Archiv f. klin. Med.*, Bd. lvi, S. 120.

² *Archiv f. Verd.*, Bd. ix, S. 262.

³ *Berlin. klin. Wochenschrift*, 1904, Nr. 25.

⁴ *Deut. med. Wochenschrift*, 1907, Nr. 33, S. 135.

Symptoms.—An important symptom is the extreme emaciation, a loss of from ten to fifteen pounds being not an unusual occurrence. This loss of flesh is a most important sign, especially since it appears without the slightest tendency to any disturbance of motility of the stomach. The appetite and quantity of food taken may be normal; occasionally, however, both may be decreased. The great loss in weight must be dependent upon the loss of gastric secretion. Amylolytic is extensively diminished and constipation is very marked. The subjective signs are of much the same character as those found in nervous dyspepsia. Patients complain of various sensations in the stomach, pressure, fulness, eructations, excessive flow of saliva; occasionally violent pains; very rarely, if ever, heartburn. Nausea or vomiting occurs exceptionally. Marked splashing is observed in the region of the stomach; tender spots painful to pressure are, however, not observed; the abdomen is occasionally distended with gas. A diminution in the chlorides and an increase in indican are frequently observed in the urine. This disease is much like atony, and Boas believes that there can be no question but that it has in many instances been mistaken for atony or nervous dyspepsia. In order to determine the presence of this condition, Boas advises a dry test meal. Inasmuch as bread contains 35.5 per cent. of water, he uses Albert cakes containing but 8.9 per cent. of water. If five Albert cakes are given as a test meal and removed from the stomach at the end of an hour under normal conditions, but a small residue will be obtained. In digestive gastrosuccorrhœa, however, 100 to 200 cc. of fluid will be obtained. On standing it forms into two layers, a lower layer slight in amount precipitated to the bottom, and above it a clear or slightly cloudy fluid three to four times as large in amount. On examination the secretion generally shows a total acidity and percentage of free hydrochloric acid usually not above and often below normal.

Boas found in a large proportion of cases a total acidity of from 40 to 56 and free hydrochloric acid 35 to 45, which indicates that while in this condition there may be no excess in the acids of the gastric secretion, the secretion is greatly increased in quantity. In other words, there is a hypersecretion but no hyperacidity. The average specific gravity of the gastric juice is 1012. The secretion presents a more or less marked biuret and sugar reaction, while albumin is not present; erythrodextrin, amidulin, and also the amylum reactions are usually marked. By means of the dry diet the diagnosis is made positive. If the bottom layer of the secretion is large, there must be a motor disturbance present at the same time. Boas also investigated the contents of the stomach in the fasting state, and pointed out three distinct types of digestive hypersecretion:

- I. Digestive gastrosuccorrhœa in which the motor function of the stomach is normal and in which no contents are obtained from the fasting stomach.
- II. That type in which more or less contents are obtained from the fasting stomach, known as permanent gastrosuccorrhœa.
- III. A type in which the motor function of the stomach is markedly disturbed at the same time.

Diagnosis.—The symptoms do not wholly point to the condition; the most marked ones are those of chronic nervous dyspepsia with marked emaciation, succussion in the region of the stomach a considerable period of time after meals and after small meals. The symptoms often point to atony,

therefore, in order to make an exact diagnosis, three forms of examinations must be used:

I. Examination of the fasting stomach.

II. Examination of the gastric contents one hour after the dry test meal.

III. Examination of the gastric contents one hour after a test breakfast.

Prognosis.—This is not unfavorable. In most instances it was possible for Boas to subdue the subjective symptoms to increase the body weight and to reduce in a measure the abnormal quantity of gastric secretion.

Treatment.—The therapeutic indications are to increase the body weight, to avoid foods that increase the quantity of gastric secretion, and, finally, to counteract the imperfect starchy digestion, all three indications being corrected by the diet. It is also necessary to overcome the constipation, and this can be accomplished by the same means, namely, by a proteid-fat and sugar diet. In place of sugar, dextrinized food can be utilized as bread; the crust of wheat bread is to be preferred. Fluids should be given plentifully in order to avoid the irritation of the gastric juice upon the mucous membrane of the stomach. Alkaline drinks should be taken plentifully during meals. Soups, alcohol, black coffee, and tea should be avoided. Excellent results are obtained by a rest cure of some weeks. Of medicinal remedies, belladonna and atropine may be utilized; on the other hand, alkalies are most beneficial, and Boas recommends especially sodium citrate (a teaspoonful four times daily), which acts better than sodium bicarbonate. This remedy may be combined with calcined magnesia and may be administered for a long period of time.

GASTROMYXORRHOEA.

By gastromyxorrhœa is meant a flow of mucus from the stomach. While various observers as Ewald, Riegel, Martius, have shown that small quantities, of mucus were occasionally found in the fasting stomach, but little attention was paid to this condition until Dauber¹ pointed out that under abnormal conditions considerable quantities of mucus were secreted by the stomach and found in the fasting state. He reported a case of Leube's in which this condition was observed, and called the disease chronic hypersecretion of mucus or gastrosuccorrhœa mucosa. Kuttner² investigated the subject farther, and found that the condition exists not infrequently, and is often observed in certain patients if lavage is practised on the fasting stomach. As has been shown by Schmidt³ and Tellerling,⁴ the appearance of small quantities of mucus in the fasting stomach may be regarded as normal; it is important, therefore, to fix a limit to the quantity to be considered normal, and any amount beyond this must be looked upon as pathological. Kuttner regards the recovery of more than 5 cc. of mucus from the fasting stomach pathological, but considers the condition one of gastromyxorrhœa when more than 25 cc. are obtained. The condition can only be considered present if the secretion of mucus is constant or reappears at certain intervals. It is important, however, to consider the method by which the mucus is obtained.

¹ *Archiv f. Verdauungskr.*, Bd. ii, S. 167.

² *Berlin. klin. Wochenschrift*, October 30, 1905.

³ *Deut. Archiv f. klin. Med.*, 1896, 3.

⁴ *Inaug. Dis.*, Bonn, 1894.

The patient should be accustomed to the use of the tube, as otherwise the mixture of saliva with the mucus will be so great as to vitiate the results. The method of moving the tube to and fro to induce the expulsion of mucus and the long continuance of the examination will increase the quantity of mucus. In order to arrive at a definite decision that there is an abnormal secretion, repeated examinations should be made. It is perfectly possible, too, that by swallowing saliva and pharyngeal mucus, secretion in the stomach may be excited; on the other hand, there must be a pathological disposition to secrete mucus in such cases, as the mucous membrane of the stomach would hardly react thus to so slight an irritation. Kuttner concludes that an increased secretion of mucus occurs in the fasting stomach, in gastromyxorrhœa. We can therefore constantly or repeatedly at intervals easily remove large quantities of mucus from the fasting stomach. This secretion has the following properties in uncomplicated cases of gastromyxorrhœa:

1. It is grayish white, milky, slimy, and stringy. It contains translucent swollen lumps or fine specks of mucus, and is without any special odor. On pouring from one glass to another, the contents are markedly stringy, and standing in a conical glass separate into two layers, a turbid fluid, the chief part, and a small layer at the bottom consisting of a whitish sediment enveloping finer and larger chumps and specks, with masses of mucus. These particles come from the bronchial, pharyngeal, and œsophageal mucous membrane, and are pigmented or even mixed with pus. Sometimes masses of mucus of a translucent, foamy, or purulent nature coming from the mouth or pharynx, float on the surface of the liquid. In some cases the mucus from the fasting stomach is colored yellowish or greenish yellow, on account of a mixture with bile or intestinal secretion. In uncomplicated cases remaining particles of food are never present.

2. The secretion in typical cases of excessive mucous secretion appeared feebly acid, neutral, or often alkaline. The total acidity varied between 6 and 8; free and combined hydrochloric acid could never be detected, nor was lactic acid present.

3. The pepsin varied by the method of Mett between 2 and 4 mm., and occasionally the ferment was completely absent; at times markedly diminished.

4. The test for sulphocyanates was negative, and also Trommer's test. On the other hand, the biuret test gave a positive reaction. Gmelin's reaction was sometimes positive, but more frequently negative.

5. When grape sugar was added to the secretion and the mixture placed in the incubator at 37° C. no formation of gas appeared even after standing several days.

6. The examination for blood was generally negative, but occasionally positive.

7. The specific gravity of the mixture varied between 1003 and 1010.

8. Determination of the freezing point gave values between 0.29° and 0.44°.

9. The microscopic examination of the secretion obtained from the fasting stomach showed leukocytes and the nuclei of leukocytes, and more rarely some of the constituents of the gastric mucosa. Occasionally yeast cells were found lying singly, and also different microorganisms. Sarcinæ or budding yeast were never present. In addition to these microscopic objects coming from the stomach, pigmented alveolar epithelium from the bronchial

mucous membrane and pavement epithelium from the mucous membrane of the mouth, pharynx, and œsophagus could be seen.

Intermittent Gastromyorrhœa.—This occurs rarely. Many cases are probably not recognized, as the discomfort is not lasting and the attention of the physician is not called to the condition. In this the attacks of increased secretion of mucus occur at intervals, repeating themselves irregularly.

Etiology.—Kuttner was unable to find any evidences of locomotor ataxia in the patients whom he investigated. In 2 the attack took its onset with disturbances in the secretions of the nose. He believes, therefore, that there is connection between the affection of the nose and that of the stomach; the mucus of the stomach, however, is not simply swallowed, but in large part due to secretion.

Symptoms.—The attack may begin with prodromal symptoms, headache, loss of appetite, nausea, etc., but more usually in the midst of perfect health, often early in the morning soon after awakening.

The most striking symptom of this paroxysmal attack is severe vomiting, which is intractable in the true sense of the word, as it cannot be successfully checked by any remedy. The vomited matter consists of large quantities of tough, slimy liquid, finally of bile and intestinal juices, but contains no food remains. In one of Kuttner's patients vomiting of bloody fluid occurred at the height of the attack. Pain in the region of the stomach is not present, or if so it is of no moment. During the attack the stomach refuses to retain food, drink, or medicine. The patient feels wretched, the abdomen is retracted, the pulse small, the tongue dry, and the quantity of urine much diminished. The attack may extend over a period of one day, or may continue from three to five or even twelve days. The attack usually ceases quite suddenly, and after the cessation of the vomiting the patient can again take food without any annoyance. The patient enjoys good health during the interval of the attacks, and often has no or slight dyspeptic symptoms. The examination of the gastric contents after a test breakfast in the interval generally shows a normal percentage of hydrochloric acid and only a slight amount of mucus.

Treatment.—In treating this condition a distinction must be made between the attack and the patient's general condition. In the beginning of the attack thorough lavage may terminate the symptoms at once, although when they are at their height lavage is valueless, and but little is to be gained by the internal administration of medicine. A hypodermic injection of morphine with atropine is sometimes useful. A condition of collapse accompanying a severe attack may be so serious, especially in patients with a weak heart, as to demand subcutaneous injections of camphor, enemata of normal salt solution, or a subcutaneous infusion. Treatment of the underlying condition is mainly directed to the removal of the neurotic tendency. At times a change of climate and atmosphere, hydrotherapy, massage, electricity, iron, and arsenic may produce a favorable influence upon the disease. Search should be made for a possible cause, either in the stomach itself or in other organs, such as the nose, and treatment should be directed to these conditions.

Continuous Gastromyorrhœa.—The chronic or continuous form is more frequent than the intermittent.

Etiology.—This condition may be associated with hypersecretion of acid or with various organic diseases, or may exist as an independent functional neurosis. Kuttner has found it associated most frequently with chronic

catarrh, as well as with those nervous affections of the gastric mucosa in which there is a marked diminution or entire absence of hydrochloric acid. He also observed it in typical cases of gastric ulcer with or without pyloric obstruction. These diseases seem to be rather favored by the ingestion of starchy food. The seat of origin of the mucus is not known, but it is probably a mixture of the secretion of the superficial epithelium and of the mucous glands, coming in largest measure from the glands of the pylorus.

It is impossible to determine whether this condition is always accompanied by a definite lesion of the mucous membrane, but as it is frequently associated with hypersecretion of acid, it is evident that, like the latter, it may depend purely upon nervous influences. Four cases of this disease have come under the writer's observation, three in females, aged thirty-two, forty-one, and forty-four respectively. Only one case occurred in a man, aged thirty-nine years.

Symptoms.—There are no characteristic symptoms, and the condition may be discovered in some cases in which there are no subjective symptoms. The diagnosis is established by the discovery of large quantities of mucus in the fasting stomach, shown to be the product of the gastric mucous membrane and not to come from other sources, such as the nose, mouth, or pharynx. It is immaterial whether or not subjective symptoms are present.

Treatment.—Lavage should be practised for the removal of the mucus, provided there is no contra-indication to the procedure; instead of this method of treatment, mineral waters may be utilized, being chosen with reference to the nature of the associated disease, the general condition of the patient, and the various gastric functions.

BULIMIA.

Bulimia is that condition characterized by an abnormal increase in the sensation of hunger. It has also been termed hyperorexia, cynorexia, and lycorexia. Rosenthal believes this disorder is due to an excitation of the hunger centre, and this is probably true in cases of the affection following brain diseases. Stiller considers it due to irritation of the peripheral nerve endings in the stomach itself. It is probable that there are some forms of bulimia due to lesions of the centre, while others are due to excitation of the nerves in the stomach itself. It has been stated that the sensation of hunger is due to stimulation of the hunger centre in the medulla by blood impoverished in the nutritive substances, and that this condition is overcome when the nutritive value of the blood is increased. That this fact will not entirely account for the sensation is shown by the statement that the appetite is satisfied as soon as the stomach is filled with food, long before the nutritive material has reached the centres of the brain. On the other hand, Leo has stated that increased appetite is due to the rapid evacuation of the stomach, which must be accompanied by a pyloric insufficiency; yet Ewald and Fleischer have observed cases in which an increased motility of the stomach did not exist. Recently, Knapp has advanced the theory that hunger is produced by contractions of the muscularis of the pyloric portion of the stomach: "As the contraction of the muscularis is the result of irritation of the mucosa, consequently the irritation of the mucosa at any time will produce contraction, which will give rise to the sensation of hunger, and as soon as the contraction gives way to relaxation, the sensation of hunger disappears."

Etiology.—Bulimia may exist as a primary neurosis or may be secondary to diseases of the stomach or other organs. As a primary neurosis, it is found in neurasthenia and hysteria. It occurs in cerebral tumors, Basedow's disease, pulmonary tuberculosis, diabetes, syphilis, intestinal worms, in ulcer and dilatation of the stomach, and in many other conditions. Bulimia is most frequently observed in females between the fifteenth and fortieth years. In the series of 1592 cases of gastric neuroses, bulimia occurred in 24 cases (1.5 per cent.). The following table represents the number of cases in males and females:

Years.	Males.	Females.
10 to 20	1	3
20 to 30	2	6
30 to 40	1	8
40 to 50	0	2
50 to 60	1	0
60 to 70	0	0

Symptoms.—This condition usually occurs in attacks at irregular intervals, although at times they may appear periodically and extend over a long period of time or be of short duration and be followed by an attack of anorexia. The disorder comes on as a violent sensation of hunger occurring after meals, in consequence of which the patient becomes exhausted, feels faint, has gastric pains, ringing in the ears, and suffers with vertigo and cold extremities. This continues until some food is taken. A very small quantity of food will at times overcome the sensation for a longer or shorter period of time; occasionally very large quantities must be ingested to relieve the condition. In one case of this series a quantity of food representing 6250 calories was consumed daily. In consequence of the inordinate amount of food, other disturbances of digestion are apt to follow, such as atony, gastritis, and disturbances of the intestines. The gastric contents were examined in 21 of 24 cases; normal acidity was found in 15 (72 per cent.), hyperacidity in 3 (14 per cent.), and subacidity in 3 (14 per cent.); hypermotility was present in 6, normal motility in 12, and atony in 3 cases.

Diagnosis.—Bulimia must be differentiated from acoria and polyphagia. In bulimia there is an abnormal desire for food, but the desire can be satisfied, while in acoria there is an absence of the sensation of satiation. In polyphagia or gluttony, while the appetite remains good, the feeling of satisfaction is delayed to such an extent as to create a constant desire for more food.

Prognosis.—This depends upon the cause, whether it is primary or secondary to some other disorder. The primary form due to neurasthenia or hysteria may be protracted or may disappear suddenly; when secondary, the prognosis depends upon the nature of the primary disease.

Treatment.—In treatment special attention should be directed toward the primary disorder. In those forms due to neurasthenia and hysteria the nervous system in general should receive special attention. In many cases rest, change of scene, hydrotherapy, electricity, massage, and psychotherapy are effectual means of combating this condition. The diet should be carefully regulated. Patients should be required to eat slowly and masticate the food thoroughly. Nourishment should be taken at frequent and regular intervals, such as every two hours. The food should consist largely of milk, eggs, cereals, boiled fish, the lighter meats, and green vegetables. Fried

foods and sweets should be avoided. Intra-gastric galvanization has proved effectual in some cases, while at times the intra-gastric douche has been used with some success. Of the medicines employed, the bromides have been used advantageously, the bromide of strontium in 15-grain (1 gm.) doses taken three or four times a day, being most useful. Arsenic, codeine, and opium have also been recommended.

PAROREXIA.

Parorexia is that condition characterized by a perversion of the appetite. According to the character of the perversion we have: (1) Pica, desire for articles which are not foods, such as chalk and earth. (2) Malacia, desire for spiced food, vinegar, mustard. (3) Allotriophagia, desire for disgusting substances, such as urine and fecal matter.

Pica and malacia are often observed in the same individual. Parorexia is found in neurasthenia and hysteria, occasionally in disturbances of the digestive tract, helminthiasis and gastralgia, and often in the insane. Malacia is frequently observed in chlorotic girls and pregnant women, while allotriophagia is found in the insane. In this series of 1592 cases there were 8 cases of parorexia (0.5 per cent.), 6 being in males and 2 in females. Three were from ten to twenty, 2 from twenty to thirty, 2 from thirty to forty, and 1 from forty to fifty years of age.

GASTRALGOKENOSIS (PAINFUL GASTRIC EMPTINESS).

Boas has introduced this term to indicate that neurosis characterized by the appearance of pain in the stomach, occurring when the stomach is empty, and which disappears as soon as food is taken. It may occur as a permanent neurosis, or may come on periodically. According to Boas, this condition is produced by severe contractions of the pylorus, and is relieved when food is present in the stomach.

Etiology.—This condition is found in neurasthenic individuals who are subject to worry and anxiety, or who have been leading an irregular life. Examination of the gastric functions shows normal conditions both as to secretion and motility. This affection occurred only in males in 7 of this series (0.5 per cent.); of these, 3 were between twenty and thirty, 3 between thirty and forty, and 1 between forty and fifty years.

Symptoms.—The condition manifests itself by the appearance of pain, which at times becomes intense, from one to two hours after meals. An attack may appear during the day or night, and last from fifteen to thirty minutes. The pain is immediately relieved by the ingestion of food.

Treatment.—This consists in properly looking after the underlying neurasthenia. Patients should be taught not to allow the stomach to become empty, and for this reason should be encouraged to take food every two hours. Boas has obtained good results by the use of opium.

GASTRALGIA NERVOSA.

Gastralgia nervosa is the condition characterized by periodic or spasmodic attacks of pain in the stomach, appearing independently of the ingestion

of food and not associated with any organic disease. The pains of a gastralgic type present in ulcer, cancer, dilatation, and in some forms of gastritis are accompanied by organic changes, and cannot be properly classified under the head of gastralgia nervosa.

Etiology.—Gastralgia is usually considered as a neurosis of the vagus, and only a marked increase in the condition to be described as gastric hyperæsthesia. Einhorn divides gastralgias into five varieties: (1) Of gastric origin; (2) of central origin; (3) of neurotic origin; (4) of constitutional origin; and (5) of reflex origin.

1. Gastralgias of gastric origin. In this class are found the neuralgic pains present in cancer and ulcer of the stomach, in certain forms of gastritis, and produced by adhesions from the stomach to other organs.

2. Gastralgias of central origin. In this class are the attacks of pain observed in the gastric crises of locomotor ataxia, and the gastric pains in cerebral tumors and myelitis.

3. Gastralgias of neurotic origin. This is often observed in hysteria and neurasthenia, and usually simultaneously with other symptoms of hysteria and neurasthenia.

4. Gastralgias of constitutional origin. Gastralgia may be due to some change in the blood, as in cases of infection, intoxication, etc. In this class are found the gastralgias of lead poisoning, malaria, anæmia, and gout.

5. Gastralgias of reflex origin. This form is due to disease of other organs, as the generative system, and enteroptosis.

Gastralgia is more frequent in women than in men. In this series of 1592 cases of gastric neuroses, there were 24 cases of gastralgia (1.5 per cent.), 17 being in females and 7 in males.

Symptoms.—The attacks of pain, as a rule, appear suddenly; occasionally they are preceded by premonitory symptoms, such as fulness in the stomach, nausea, vomiting, and headache. The pain may be burning, boring, tearing, cutting, or gnawing, and is usually felt in the epigastrium; at times it radiates throughout the entire abdomen or even to the back, and may be so intense as to cause weakness and collapse, the patient having a thready and irregular pulse, with pallor, cold extremities, and cold perspiration. Often the pains are relieved by pressure; at times these symptoms are accompanied by nausea, vomiting, and hysterical manifestations, such as globus hystericus and nervous chills. The duration varies, the attacks may last from a few moments to many hours, and the intervals between may be days, weeks, or months. In the gastric crises of locomotor ataxia the attacks of gastralgia are usually ushered in with pains in the legs and arms, nausea and vomiting. These crises may precede the classic signs of locomotor ataxia by months or years. The gastralgia accompanying malaria appears at the same time every day, every other day, or every third day, and, as a rule, is accompanied by chills and fever. Boas calls special attention to the gastralgic pains occurring in neurasthenic females at the menstrual period.

In most instances this condition bears no relation whatever to the character of the food taken. The pains begin in the epigastrium, remain localized, and are burning and boring in character. The attacks last from fifteen to thirty minutes, and are repeated several times during the day, while at night the pain is absent. Another form of gastralgia to which much attention has been directed is that form produced by open inguinal or femoral rings,

occasioning small or even occult hernia, also by hernia of the abdominal walls, occurring in the median line along the linea alba or laterally (in the mammary line). These are observed more frequently along the linea alba, either near the ensiform cartilage or near the umbilicus; males are more often affected than females, and more commonly those of the working classes. The pain is colicky, and ceases when the patient reclines. Neurasthenic symptoms are almost always present.

Diagnosis.—This is made by the character of the attacks with paroxysmal pain, bearing no relation to the ingestion of food, and at the same time not associated with any organic or other functional disease of the stomach. As gastralgia is usually due to some other condition, care should be taken to determine as far as possible its etiological relation. Gastralgia must be differentiated from ulcer. In ulcer there is a circumscribed painful area in the epigastrium and in the back to the left of the eleventh or twelfth dorsal vertebra. The pain is proportionate to the quality and quantity of food ingested, and is accompanied by nausea and vomiting; is relieved by vomiting; in ulcer occult blood is found in the *fæces* at times. When there is still doubt Leube recommends the ulcer rest cure in order to determine from the result of the treatment the presence or absence of ulcer. Gastralgia is differentiated from chronic gastritis by the slower appearance of the pains after the ingestion of food, by the presence of the paroxysms of pain, and by the absence of any change in the gastric secretion. It is differentiated from cancer by the absence of the usual signs; the pain usually not being paroxysmal in cancer. Intestinal colic may be mistaken for gastralgia, but in it the intestine is inflated with gas, and the pain is relieved by the passing of gas or a movement of the bowels, which frequently contain mucus. The pain of intestinal colic changes its position, while in gastralgia it is localized, as a rule, in one area. Intercostal neuralgia is distinguished from gastralgia by the fact that in the former condition the intercostal area is painful to pressure along the course of the nerve. Gallstone attacks may be mistaken for gastralgia. In these the pain is generally to the right of the median line, the attack is accompanied by fever, and there is often a sensitiveness of the liver. In some cases the diagnosis is most difficult, especially when the more important symptoms of cholelithiasis are absent. Nephritic colic is differentiated from gastralgia by examination of the urine. The pain in nephritic colic radiates toward the bladder.

Treatment.—In the treatment of gastralgia it is important to direct our attention to the underlying cause. The form due to anæmia should be treated with iron and arsenic; that due to malaria by means of quinine; and that from nicotine poisoning by cautioning against the use of tobacco. Reflex gastralgia must be handled by treating the primary disease. That due to neurasthenia and hysteria should be treated by change of scene, rest, electricity, and massage. In patients much run down a systematic rest cure will produce excellent results. The attacks of pain are treated as follows: hot applications in the shape of a hot-water bag or hot poultices or packs are placed on the abdomen and the patient given half a grain (0.03 gm.) of codeine or 20 to 30 drops (1.5 cc.) of Hoffman's anodyne. Belladonna and chloroform water have also been recommended, as well as phenacetin and antifebrin. If the attacks of pain are intense, a hypodermic injection of morphine must be administered or opium and belladonna given by rectum. As an effective means in the relief of pain, the galvanic current is of consider-

able value, the anode being placed on the epigastrium and the cathode on the spinal column. Strong currents are used. At times better results may be obtained by means of intragastric galvanization.

HYPERÆSTHESIA GASTRICA.

By gastric hyperæsthesia is meant that condition in which the mucous membrane of the stomach is unduly sensitive, even to its normal contents. The lightest forms of foods will often cause distress in spite of the fact that the stomach presents neither secretory nor motor disturbances. The character of the discomfort may be pain, fulness, burning, or sensations of cold occurring after meals. In some instances, not only does the food cause discomfort, but the normal amount of hydrochloric acid will cause pain, similar to that found in hyperacidity, and it may exist even when the acid is diminished or absent. Attention has been called to this fact by Talma, Stockton, and Steele.

Under the subjects of achylia gastrica and hyperchlorhydria, it was shown that symptoms of hyperacidity occasionally exist in achylia gastrica; in these cases there is evidently a hyperæsthesia of the gastric mucous membrane, which is so sensitive that it cannot tolerate even the normal amount of acid. Again, a condition is often observed in which the percentage of acid may be far in excess of normal, and yet the individual have perfect health without gastric disturbances. This indicates that the acid itself is not alone sufficient to produce the symptoms of hyperacidity, but that there must be in addition a hyperæsthesia of the gastric mucosa. In the cases described by Talma there was an idiosyncrasy for hydrochloric acid, the slightest quantity producing pain in the stomach; these manifestations are due to an abnormal sensitiveness.

Etiology.—As a primary affection, hyperæsthesia is found in neurasthenia and hysteria, and frequently in anæmic females; it occurs secondary to affections of various organs, such as ulcer of the stomach, chronic gastritis, and disease of the kidneys (uræmia). It may be produced by excesses in food and drink, the abuse of coffee, tea, alcohol, indulgence in highly seasoned food and tobacco, or it may come from drug habits, such as opium or cocaine. The taking of insufficient food, fasting, living on an exclusive diet, or eating irregularly are also etiological factors. Hyperæsthesia is the affection from which conditions such as anorexia nervosa, nervous nausea and vomiting, gastralgia, and sitophobia often take their origin. In this series of 1592 cases there were 31 cases of hyperæsthesia (2 per cent.).

The following table represents the number of cases in males and females arranged according to age:

Age.	Males.	Females.
10 to 20	1	12
20 to 30	1	8
30 to 40	2	2
40 to 50	0	3
50 to 60	0	2

Symptoms.—The sensation produced is that of mild pain, fulness, and burning, appearing soon after meals and continuing during digestion, and ceasing when the stomach is empty. In some cases the pain is greater, and may cause nausea and vomiting. The disturbance is often greater from liquid than from solid food. On account of the discomfort produced,

much food is discarded and the patient emaciates and loses strength. As has already been stated, some of the cases manifest the symptoms of hyperchlorhydria, notwithstanding the fact that the acidity may be normal or subnormal. In these cases the discomfort appears several hours after meals and disappears on the ingestion of food or the taking of alkalis. On palpation the whole gastric area may be sensitive to pressure but free from localized painful points. The gastric secretion shows a normal percentage of free acid, with occasional slight variations. The motor function of the stomach is not disturbed.

Diagnosis.—This is usually made with ease, provided the symptoms above described are present together with a normal gastric secretion. Hyperæsthesia must be differentiated from ulcer and chronic gastritis. In ulcer there is a localized painful area and the pain is proportionate to the quality and quantity of food ingested. In chronic gastritis there is usually a diminution of gastric secretion, together with the presence of mucus, while the symptoms of distress do not appear immediately after meals, but some time later.

Treatment.—In all instances the patient should be required to remain in bed, at first on a milk or egg albumen diet, and, if possible, combined with a systematic rest cure, the food being increased gradually, until solid food is taken. In serious cases, rectal alimentation must be practised for a few days at the onset. Cold compresses should be applied to the abdomen. Galvanization is efficacious in some cases. Nitrate of silver may be given by mouth, grain $\frac{1}{2}$ (gm. 0.008) in solution, three times daily, or it may be administered by means of the stomach douche. At times the bromides, valerianates, and sumbul prove useful, especially in those forms due to neurasthenia and hysteria.

Attention must always be directed toward the treatment of the underlying neurasthenia or hysteria. In the cases dependent upon anæmia, iron is indicated.

Gastric Idiosyncrasies and Abnormal Sensations.—There are certain individuals who manifest an idiosyncrasy toward certain foods which when taken produce gastro-intestinal disturbances or some eruption on the skin. Among the foods causing this class of symptoms are fish, berries, cheese, etc. Fever, headache, gastric pain, nausea, vomiting, and urticaria are accompanying symptoms. The writer has constantly noticed the condition in a certain patient after eating eggs. The only means of preventing it is to order the individual to abstain from the food that causes this discomfort. There are abnormal sensations met with in neurotic individuals, such as cold, heat (stomach burn), epigastric pulsations, constrictions, and sensations of a foreign body in the stomach. The gastric secretion is normal in all these instances, and there are usually other neurasthenic symptoms present that indicate the nature of the disorder. The treatment must be directed to the nervous system in general. For this purpose massage, electricity, hydrotherapy, and psychotherapy play an important role. The bromides and belladonna are at times of some value.

MERYCISM OR RUMINATION.

By merycism is meant that condition in which there is a regurgitation of food into the mouth which is again masticated and then either reswallowed

or spit out. This act is accompanied by a more or less pleasurable sensation and without nausea or effort, and occurs some time after meals. Rumination in man is comparable to the same condition found in certain animals. Some authorities, following Damur,¹ attribute this disorder to a paresis of the cardia, while others consider it a reflex neurosis causing a temporary relaxation of the cardia. Rosenthal holds it is due to an irritation of the vagus, producing an opening of the cardia through Openchowski's dilator fibers, occasioning an antiperistalsis of the œsophagus. In opposition to the view that the condition is due to paresis of the cardia is the fact that the deglutition sounds remain perfectly normal, and that, if the stomach is inflated, the air does not escape. According to Luschka and Arnold, regurgitation is produced by a dilatation of the lower part of the œsophagus. There are two classes of phenomena aiding in the production of the disorder, one active, the other passive; the active consists in depression of the diaphragm and contraction of the muscles of the abdomen, both of which produce a void in the thoracic cavity; and contraction of the abdominal muscles which compress the stomach. "The passive phenomenon consists in the elongation and widening of the œsophagus and the diminution of the lumen of the stomach" (Spivak).

Etiology.—Heredity plays a role in a number of cases which have been reported. Rossier has reported this condition in a father and two sons, and Loewe cites a family in which four members were ruminants. The writer has observed the condition in a father of fifty-one and a daughter aged fourteen years. In many cases other neurotic and hysterical tendencies have been observed. At times the condition is acquired by imitation. Koerner reports a case of this character in which a governess addicted to this habit imparted the condition to her two pupils.

Among the cases to which rumination is said to be due are rapid eating, worry, fear, nervous strain, and sudden shock. A large number of cases occur among idiots and the insane. The disease is observed more frequently among men than women. It is found at all ages, and is most common in highly intellectual men. In the series of 1592 cases of gastric neuroses there were 24 cases of merycism (1.5 per cent.). The following table represents the proportion among males and females at various ages:

Age.	Males.	Females.
10 to 20	3	1
20 to 30	2	2
30 to 40	4	0
40 to 50	6	1
50 to 60	3	0
60 to 70	2	0

Among them were five students, one artist, two lawyers, three physicians, five clergymen, two merchants, one baker, and one mechanic. According to Johannessen,² the regurgitated material shows free acid at the end of rumination. The state of the gastric secretion varies in merycism. Alt found a hyperacidity in one of his patients, Boas a subacidity in one case, and Jurgensen found no free hydrochloric acid whatever. The gastric contents were examined in 19 of the 24 cases of this series; a normal acidity existed

¹ *De la paralysie du cardia ou merycism*, Thèse, Berne, 1859.

² *Zeitschrift f. klin. Med.*, Bd. x, S. 274.

in 11, hyperacidity in 3; subacidity in 2; anacidity in 1; and heterochylia in 2 cases. Atony was found in 3 cases, while in the others the motor function was normal.

Symptoms.—Rumination usually begins as a voluntary process in an insidious manner; on account of gastric discomfort the food is first regurgitated, producing a pleasurable sensation it is again reswallowed, and finally the condition of merycism is established as an involuntary process. The regurgitation usually takes place during the early period of gastric digestion; it rarely occurs in the fasting stomach; the remastication continues as long as the regurgitated food has a pleasant taste; as soon, however, as it becomes acid (during the late period of digestion) it is either swallowed at once or ejected. Rumination may take place after every meal and last for a very short period of time or extend over hours.

Merycism occurs alone or may accompany other gastric disorders; it is occasionally associated with atony and dilatation of the stomach. Von Hacker and Singer have shown that an insufficiency of the cardia and a dilation of the lower third of the œsophagus may be due to the mechanical expansion occasioned by the regurgitation of large bits of food. As a rule, the general nutrition of patients suffering with this disorder is good; although when large portions of all meals are not reswallowed emaciation is produced and the general nutrition becomes markedly affected.

Diagnosis.—This is made, as a rule, without difficulty. It differs from regurgitation in that in the former condition the food is again masticated, while in the latter this process does not take place. In vomiting, nausea and retching are present, while merycism is accompanied by pleasurable sensations.

Prognosis.—This is often good, especially if the patient makes the effort to overcome the habit. This condition may extend over a long period of time, being present occasionally during the entire life, or it frequently disappears for a longer or shorter period, to recur again following some mental strain or shock.

Treatment.—This consists essentially in an autosuppression. In a certain number of cases the patient can be taught to overcome this disease. He should be required to eat slowly and masticate thoroughly; 3 patients of this series were cured in this way. The state of the gastric secretion occasionally gives a clue as to treatment. Hydrochloric acid is useful in cases of subacidity, while alkalis are of benefit in superacidity. Intragastric electricity has been recommended, and was of great benefit in 2 patients. In those who are much run down and weakened by disease a thoroughly regulated rest cure with psychotherapy is most effective; 3 patients were cured in this way. Strychnine and quinine have been recommended, inasmuch as their taste destroys the desire to remasticate. Lavage has been used by Johannessen and gavage by Jurgensen.

REGURGITATION.

By regurgitation is meant that condition observed in hysterical and nervous individuals when food is brought up in small quantities from the stomach into the mouth and usually ejected. This disorder is much like rumination, except that food is not remasticated; rumination, however, may develop from

long-continued regurgitation. The following case illustrates this condition: A. T., a lawyer, aged thirty-five years, has always been in good health; three years before seeking medical advice he began to regurgitate his food; this occurred usually after mental strain or excitement; at first the condition took place exceptionally; finally, it came on at times when the patient found it impossible to eject the food from his mouth. He then began to ruminate, and has done so ever since. On examination, no organic disease was discovered and the gastric contents were found normal.

Etiology.—This is the same as that of rumination. The condition is usually found in neurasthenic and hysterical individuals, and is at times voluntary; however, as the habit becomes established it becomes involuntary. Regurgitation may be occasioned by a nervous strain or shock, or may be secondary to some other gastric disturbance, such as dilatation, catarrh, or hyperchlorhydria. This disorder is also largely found in adult males, and more frequently in individuals pursuing intellectual pursuits. It occurs more frequently than rumination. In the series of 1592 cases of gastric neuroses it was present in 32 cases (2 per cent.), 26 being in males and 6 in females. As to age, 2 were in the second decade, 10 in the third, 7 in the fourth, 9 in the fifth, 3 in the sixth, and 1 in the seventh.

Among those suffering from this affection, there were two artists, three students, three physicians, six lawyers, five ministers, four teachers, three merchants, one mechanic, and one laborer. The gastric secretion is usually normal. In the series of 32 cases the gastric contents were examined in 29, and normal acidity was found in 23 (79 per cent.), hyperacidity in 3 (10 per cent.), subacidity in 2 (8 per cent.), and heterochylia in 1 (3 per cent.). The motor function was usually normal, a slight atony existing in five.

Symptoms.—The onset is gradual, and consists in the regurgitation of food from the stomach soon after eating and persisting during the entire period of digestion. It is not preceded or accompanied by nausea, and if it occurs early in the process of digestion, the regurgitated material has the same taste as the food swallowed. Later it becomes sour from the mixture of the acid of the gastric juice. At times this process can be suppressed, at other times not. The food is not remasticated and swallowed with pleasure, as in the cases of rumination. Regurgitation may occur after any meal, or only after certain ones; it may disappear for a time, to recur after some mental strain or excitement. When large portions of all meals are habitually regurgitated and ejected, the general nutrition of the patient is likely to suffer, and he may become materially emaciated and anæmic.

Diagnosis.—Regurgitation is differentiated from vomiting by the absence of the usual signs of the latter, nausea, retching, salivation, etc. It is distinguished from œsophageal regurgitation due to stenosis of the œsophagus by the presence of the gastric secretion in the regurgitated matter. The difficulty or inability to pass the tube in œsophageal stenosis aids in differentiating this from gastric regurgitation.

Prognosis.—This is usually favorable, provided the patient will assist in the act of suppression of the condition; the health of the patient becomes seriously affected only in those instances in which all food is constantly regurgitated.

Treatment.—The general condition of the patient should be looked into and the associated neurasthenia or hysteria treated by appropriate means. An important factor in treatment consists in teaching the patient voluntary

suppression. In emaciated individuals this form of treatment may be assisted by a rest cure with isolation. This treatment proved highly satisfactory with three of this series. The patient should be required to eat slowly and masticate his food thoroughly. Intragastric electricity is valuable in some cases. Strychnine and the bromides have been used with good results.

ERUCTATIO NERVOSA.

Eructatio nervosa is a condition characterized by periodic or paroxysmal attacks of noisy belching; it occurs in neurasthenic and hysterical individuals. The question of the origin of the gas expelled has attracted much attention. The following sources have been suggested: the fermentation or putrefaction of food, causing the formation of gases, or the swallowing of air (aërophagia). It is now generally admitted that the gas which is expelled in eructatio nervosa is swallowed, and that the air passes into the stomach by an act of deglutition. The swallowing of small quantities of air is a natural phenomenon accompanying the deglutition of food. Aërophagia, on the other hand, occurs as a voluntary act, induced to relieve an uncomfortable sensation in the stomach or œsophagus by the expulsion of gas that has been swallowed. Aubert¹ considers aërophagia a voluntary act, the mechanism of which is similar to that of swallowing food, while Bouveret² ascribes it to a clonic spasm of the pharynx and divides the eructation into two acts, the deglutition and the expulsion of air. Convulsive deglutition is accompanied by forcing air into the œsophagus and stomach, while expulsion is occasioned by contraction of the œsophagus expelling the accumulated air with a loud characteristic sound.

Oser³ ascribes this condition to aspiration, air being suctioned in and expelled through the œsophagus by expansion and contraction of the stomach. Linossier believes that aërophagia is much like regurgitation, and that while in regurgitation there is an expulsion of food, in aërophagia there is an expulsion of gas. In some cases, as Ewald points out, the belching has no connection with the stomach, but originates from the œsophagus by contractions of the muscles of the neck.

Etiology.—Eructatio nervosa is usually found in hysterical or neurasthenic individuals. It is more common in females, and in younger than in older persons. In the series of 1592 cases of gastric neuroses there were 54 cases of eructatio nervosa (4 per cent.), 42 in females and 12 in males. As to age, 12 were under twenty, 15 in the third decade, 15 in the fourth, 8 in the fifth, 3 in the sixth, and 1 in the seventh.

While this condition is usually a simple gastric neurosis produced by some mental excitement, such as anger, worry, or great sorrow, yet it may occur as a result of some other gastric disturbance. Catarrh, gastroptosis, or a reflex condition secondary to disturbances of other organs, such as the genito-urinary organs and heart, may produce it.

Symptoms.—This disorder usually develops suddenly and is accompanied by noisy eructations, which vary in duration and intensity, the paroxysm lasting from a few hours to days. Frequently there is a period of quiescence

¹ *Lyon Médical*, 1891, lxxvii, pp. 463, 510, 547.

² *Revue de Méd.*, 1891, 148.

³ *Die Neurosen des Magens*, 1885, S. 137.

between the paroxysms, the attack presenting no definite relation to the quality and quantity of food taken. The number of eructations varies greatly. In the case of Cortelliere,¹ 2500 eructations were produced in one hour. Spivak² calls attention to two forms of nervous eructations, voluntary and involuntary. In the first variety the patient controls the condition, and is able to inhibit this habit at will; in the second, which generally occurs during an attack of hysteria, he is unable to do so. The attack is spasmodic in character and cannot be controlled. The paroxysm usually disappears suddenly and ceases during sleep. On examination, patients suffering with nervous eructations present a rather increased sensibility to pressure in the epigastric region. These areas on pressure occasion eructations (aërophagenc points, Bandonin). Examination of the gastric contents may or may not reveal normal conditions. In this series of 54 cases the gastric contents were examined in 51; normal acidity existed in 42 cases (82 per cent.), superacidity in 6 (12 per cent.), and subacidity in 3 (6 per cent.).

Diagnosis.—This is not usually difficult. The characteristic paroxysmal attacks can scarcely be mistaken. There is usually an absence of organic disease; the gastric contents are frequently normal (82 per cent. of this series); the gas expelled was atmospheric air and showed no signs of fermentation, while the examination of the patient usually revealed other neurasthenic or hysterical symptoms.

Treatment.—Mild forms of the disorder may be combated by explaining its nature to the patient and urging him to control the attack. Sometimes, however, this is difficult, and we must content ourselves with the treatment of the neurasthenic condition. This may be accomplished by hydrotherapeutic measures, electricity, and change of scene. In persons who are much run down and have lost much flesh nothing will accomplish such excellent results as a well-regulated rest cure. At times good results may be obtained by means of lavage, douching the stomach, or by means of intragastric electricity. Drugs are usually of little value. Among those employed in this condition with more or less effect are the bromides, chloral, codeine, and belladonna.

NERVOUS VOMITING (VOMITUS NERVOSUS).

The act of vomiting is a very complicated process; not only does the nervous system play a part in the act, but also the muscles that govern the movements of the stomach. In addition to the centres governing the stomach movements there is a coördinated action of the vomiting centre from which this act takes its origin. The vomiting centre is in the medulla in the vagus nucleus in close proximity to the centres transmitting impulses to the muscles of the stomach, diaphragm, and pharynx. During the act of vomiting the abdominal muscles and diaphragm contract, the pylorus is closed, and the cardia opened, while the stomach contracts and throws its contents into the œsophagus. The œsophagus is shortened and widened by the contraction of the longitudinal fibers, the epiglottis closes and shuts off the larynx, while the soft palate closes the nasopharynx. The contents of the stomach are forced into the mouth by the antiperistaltic contraction of the œsophagus.

¹ *Wiener Allg. med. Zeit.*, 1885, S. 3.

² *New York Medical Record*, April 29, 1905.

Etiology.—Vomiting may be due to one of three conditions: to some abnormal state of the food, to some disease of the stomach itself, or to some disturbance of the nervous system. The form with which we are here concerned comes under the latter head, and is termed nervous vomiting. It may be divided into three groups:

1. Cerebrospinal vomiting due to a lesion or functional disturbance of the nervous centres.

2. Nervous vomiting proper, or vomiting due to neurasthenia or hysteria.

3. Reflex vomiting.

Stiller¹ points out the following features as characteristic of this form: (a) The ease of the vomiting. (b) Its non-dependence upon the quality and quantity of the ingested food. (c) The capriciousness with which very bizarre articles of food are retained to the exclusion of others. (d) The occasional elective vomiting that consists in selecting only one form of food which is separated from the chyme. (e) The case with which patients bear this condition even for a long period of time. (f) The very slight degree of inanition produced by the habitual vomiting. (g) The extraordinary influence of the slightest external or internal causes that react on the patient's temperament. (h) The occurrence of vomiting frequently, even on a fasting stomach, and the appearance of this condition independently of the meal. (i) The presence of other nervous symptoms associated with the vomiting or alternating with it.

In this series of 1592 cases of gastric neuroses there were 49 of nervous vomiting (3 per cent.), 34 in females and 15 in males; as to age, 6 were in the first decade, 6 in the second, 14 in the third, 11 in the fourth 8 in the fifth, 3 in the sixth, and 1 in the seventh.

Divided into the groups already described, the cases may be represented as follows:

	Males	Females	Total.	Per cent.
1. Cerebrospinal vomiting	5	3	8	17
2. Nervous vomiting	4	19	23	48
4. Reflex vomiting	6	12	18	35
Total	15	34	49	100

1. Cerebrospinal Vomiting.—The cause of this form of vomiting (also known as central vomiting) is found in the brain, spinal cord, or the membranes covering these structures. The condition has been observed as a symptom of inflammatory disease of these structures. It is found in meningitis, encephalitis, apoplexy, abscess, cerebral tumors, not infrequently after brain anæmia or hyperæmia, concussion, intoxication from ether, chloroform, opium, tobacco, or septicæmia and auto-intoxication. Spinal vomiting due to disease of the cord is rather infrequent; it occurs most commonly in the gastric crises of locomotor ataxia and occasionally in transverse myelitis. The gastric crises are characterized by the occurrence in the midst of well-being, of sudden attacks of intense pain appearing in the epigastrium radiating toward the sides and back. They are followed by the vomiting of large quantities of food, mucus, bile, and finally of intestinal juices and occasionally of blood. After copious vomiting temporary relief is afforded, and the attack ceases, especially during the night, to return again

¹ *Die nervose Mogenkrank*, 1884, S. 151.

on the following day. It may continue at intervals from six to eight days, or may last even longer. The vomiting may cease as suddenly as it appeared, the appetite may return, and the general condition of the patient improve.

During an attack the patient becomes weak and prostrated, the pulse is feeble and rapid, the appetite disappears, the bowels become constipated and the thirst intense, while the abdomen is much retracted. The examination of the vomited matter for free hydrochloric acid may reveal a normal, subnormal, or hyperacid secretion. The diagnosis of locomotor ataxia is made by the presence of the other characteristics of this disease, but inasmuch as gastric crises may occur as early symptoms of tabes, the diagnosis may be difficult. In all doubtful cases the pupillary and patellar reflexes, as well as the other symptoms of locomotor ataxia, must be carefully inquired into.

2. Nervous Vomiting Proper.—This form of nervous vomiting is most frequently associated with neurasthenia or hysteria. It is found mainly in hysterical females; in this series it occurred in 19 females to 4 males. The vomiting is often accompanied by pains in the stomach, and comes on more or less frequently every day or every few days and is often observed after some severe mental strain or excitement. The nausea that usually accompanies vomiting is frequently absent in these cases, the vomiting occurring without any effort whatever; other symptoms of neurasthenia or hysteria are either associated with the vomiting or alternate with it. The *diagnosis* of nervous vomiting is established by the presence of other associated nervous symptoms, and by its non-dependence on the quantity and quality of food taken; further by its irregularity, the expulsion of either only solids, or only liquids, or only one particular form of food, or only a portion of the meal, and the very slight loss in strength and weight. In this condition the skin is usually dry; the quantity of urine excreted is small, with a marked diminution in urea. The vomiting may continue for days or months and then disappear very suddenly without apparent cause.

Periodical Vomiting.—This condition was first described by von Leyden, and is characterized by vomiting occurring at regular intervals; between the attacks the patient is in normal health, and complains neither of gastric distress nor disturbance of the nervous system. The attacks come on abruptly and disappear as suddenly, lasting from a few hours to twelve or fourteen days, the attack as well as the interval having a constant and definite duration in every case. The attack is usually ushered in abruptly by nausea, gastralgic pain, and headache, followed by vomiting, at first of gastric contents and mucus, and then of bile. The vomiting is excessive, no food, not even fluids, being retained by the stomach during this period. The patient becomes weakened and prostrated, the pulse weak, the tongue dry, severe lancinating pains often occur in the extremities, constipation becomes marked, and the urine is diminished. The vomiting ceases suddenly and the attack gradually passes off, to be repeated again at an interval of from two to four or six weeks or even several months. When the attacks occur at short intervals emaciation may be extreme, and the disease becomes most serious, in consequence of the extreme inanition produced. A distinctive feature of the disorder is its typical periodicity, by which it can be distinguished from the gastric crises of tabes. An examination of the stomach contents in these cases usually reveals normal conditions.

Psychic Vomiting.—This form of nervous vomiting is generally due to some form of shock, fright, or sudden mishap. It may be mild or intense. In mild forms only portions of the food are vomited for a certain period of time, and the attack then disappears, the general nutrition not being markedly impaired; in the severe form, however, the inanition due to the insufficient retention of food may be so great that the disease may become extremely serious, or even fatal.

Juvenile Vomiting.—This form of vomiting occurs in young school children who have been subjected to overstudy. In addition to the vomiting, other symptoms, as a rule, present themselves. The disorder usually begins with gastric pains and vomiting, to which such symptoms as headache, pallor, dilated pupils, a slowing of the pulse, and constipation are added. In these cases improvement always follows when the patient is allowed to discontinue his school work.

Periodic Vomiting in Children.—A peculiar form of periodic vomiting, differing from the form described as juvenile vomiting, occurs in young children. It comes on usually at the age of from two to three years, and generally passes off as the child becomes a few years older. The condition has been fully described by Snow, Griffith, and others. The attack is characterized by a sudden onset, in the midst of good health, of intense vomiting, accompanied by fever and great depression, that ceases as suddenly as it began. In a certain number of cases the attacks appear with a distinct periodicity at intervals of every few weeks or even months. In at least some Snow has shown that the gastric irritability is due to an intermittent hyperchlorhydria, a secretory gastric neurosis. The cause of this recurrent vomiting is unknown, but it is probably a transitory auto-intoxication that affects the nerve centres governing gastric secretion and motion.

3. Reflex Vomiting.—This is a very common form of nervous vomiting, and may be produced by various diseases affecting the stomach and other organs. It is found as one of the symptoms of disease of the middle ear, disturbances of the eye, pharynx, and affections of the larynx. To these may be added diseases of the lungs and abdominal organs, *i. e.*, the stomach, intestines, liver, kidneys, male and female generative organs. Reflex vomiting is also common during pregnancy.

Gould¹ and others have pointed out the frequent dependence of this as well as other gastric neuroses upon eyestrain, while Brav² has found astigmatism a common cause of vomiting in school children. The vomiting of pregnancy is often a reflex neurosis of the sympathetic nerves. It is produced by tension of these nerve fibers prevented from stretching by inflammatory processes, and is often occasioned by adhesions between the posterior wall of the uterus and the promontory of the sacrum or by a rigid os or malformations of the uterus. Evidence of the existence of this condition is shown by the relief of the vomiting following correction of the abnormality. In another form vomiting is due to some hysterical or neurasthenic tendency. In these cases, as Williams points out, the neurotic nature is indicated by the fact that a cure frequently follows the employment of perfectly useless and unphysiological procedures. Many of these cases, therefore, may be cured by rest or simple suggestive means. A third variety of vomiting of

¹ *Journal of the American Medical Association*, March 24, 1907.

² *New York Medical Journal*, August 26, 1905.

pregnancy is the toxæmic form, which is a very serious disease and is produced by a marked disturbance of metabolism. Williams¹ shows that the exact origin of the condition has not been established. It is known, however, that it usually ends in death, the patient presenting signs of profound intoxication, even dying in coma. In the last stages of the disease the vomited matter is coffee-grounds in appearance and is expelled without effort; the urine is diminished in quantity and does not contain albumin or casts except shortly before death. It presents, however, a decided decrease in the total amount of nitrogen excreted, the proportion of urea is diminished, while there is a marked increase in the ammonia output and the amido-acids are increased. At autopsy lesions are found in the liver and kidneys.

Diagnosis.—The diagnosis between the various forms of nervous vomiting is not always a simple matter; after establishing the nervous origin of the vomiting it is most important to determine its exact cause, whether, for example, it is reflex or due purely to neurasthenia. These facts, as a rule, can be elicited by a complete investigation into the clinical history of the case and by a rigid examination of the patient. A careful inquiry into the various characteristic features of nervous vomiting as described by Stiller will assist.

Prognosis.—This depends largely upon the cause. In the cerebrosplinal vomiting it is usually bad, although in the gastric crises the vomiting may abate entirely, notwithstanding the continued progress of the disease. In the form associated with neurasthenia or hysteria the prognosis is usually favorable when under proper treatment, but occasionally a fatal outcome is met with, due to exhaustion occasioned by the continued and prolonged vomiting. Three cases of this character have come under the observation of the writer. In the reflex form the prognosis is favorable provided the cause can be removed.

Treatment.—The chief aim is removal of the cause. Inasmuch as this is not always possible, many patients must be treated symptomatically. In the milder forms, change of scene, avoidance of mental excitement, and rest will overcome this condition. In severe cases patients should always be placed in bed at a hospital under rigid isolation and rest cure treatment. In seven of this series the writer is convinced that the lives of the patients were saved by this plan of treatment. The very rapid recoveries that are made by patients after entering the hospital are convincing evidence of the truth of this statement.

As regards diet, the patient should be fed on liquid food, given in very small quantities, such as champagne, ice-cold milk, clam broth, or cold egg albumen. In severe forms the patient must be fed for some days exclusively by the rectum and the thirst relieved by saline enemata. In some cases solid foods are well borne when liquids cannot be taken. These should be given in very small quantities; scraped beef, eggs, rice, and toast are especially useful. Hydrotherapy may be practised with good results in some cases; this must be varied according to the conditions of the individual. Electricity has a beneficial effect in some instances, the galvanic current being used with the anode in the stomach and the cathode on the spinal column. The writer has had good results at times from lavage with solutions of nitrate of silver, at other times with solutions of menthol. As to the drugs that are used with more or less effect in the treatment of this condition, the following may be men-

¹ *American Journal of the Medical Sciences*, September, 1906.

tioned: Morphine given hypodermically is valuable in many cases, oxalate of cerium, cocaine, menthol, the bromides, chloral, validol, and orthoform. Boas recommends the iodide of potassium and bromide of sodium in the gastric crises, and rectal suppositories of opium and belladonna are useful in attacks of periodic vomiting. In the periodic vomiting of children Rachford suggests the use of calomel and bicarbonate of soda, followed by a saline laxative, and then benzoate of soda in from 3 to 8 grain (0.2 to 0.5 gm.) doses every two or three hours dissolved in essence of pepsin and peppermint water. Snow also recommends an alkaline treatment in these cases. For the vomiting of pregnancy which has become obstinate and excessive, the patient should be put to bed for a time and given ingluvin, the bromides, the oxalate of cerium, or anæsthesin; when all other measures prove fruitless the uterus should be emptied.

NAUSEA NERVOSA.

Nausea is observed in many gastric affections, as well as in diseases of other organs. There is, however, a purely functional form of nausea which is entirely of nervous origin and is due to hysteria or neurasthenia. According to Boas nervous nausea arises from a "condition of changing blood circulation in the brain."

Etiology.—Nausea nervosa is often found in anæmic and chlorotic females; it is also observed with menstrual disturbances and frequently at the menopause. It begins, as a rule, after great excitement, worry, or anxiety in neurasthenic or hysterical individuals. This disorder is most often observed in women and rarely in men. According to Boas, it usually follows chlorosis or anæmia in females. In the series of 1592 cases of gastric neuroses there were 40 cases of nausea nervosa (2.5 per cent.), 30 in females and 10 in males. As to age, 13 were in the second decade, 9 in the third, 7 in the fourth, 7 in the fifth, and 1 in the sixth.

As a result of examination of the gastric contents, in 34 cases there was a normal acidity and in 6 a hyperacidity. The hyperacidity was in all instances accompanied by anæmia. The gastric motility was normal in all.

Symptoms.—Nausea may be intermittent or continuous. In the intermittent form it appears any time of the day or night and bears no relation whatsoever to the ingestion of food, its quantity or quality. In the continuous form it persists for days and months in varying degrees of severity, sometimes slight, at other times so intense as to cause vomiting. The nausea is not dependent upon the quantity or quality of the food ingested, yet it causes a loss of appetite, and the patients, as a rule, live upon a very limited diet, the result being great emaciation.

Diagnosis.—All organic diseases accompanied by nausea must be excluded. The most important of these are incipient tuberculosis, diseases of the male and female genital organs, arteriosclerosis, uræmia, and organic diseases of the stomach.

Treatment.—In many cases due to anæmia or chlorosis appropriate treatment will relieve the condition. Attention should be paid to repair of the nervous system, especially by massage, electricity, and hydrotherapy. In those cases accompanied by marked anæmia and great loss of flesh, splendid results are obtained through a well-conducted rest cure. The

complete isolation of the patient is always necessary in severe forms. Relief is shown in some cases by means of the bromides, valerian, sumbul, and chloral. Validol for milder forms has been of great service in the writer's experience.

CARDIOSPASM.

Cardiospasm is characterized by a spasmodic contraction of the cardia, not due to any organic disease. Under normal conditions there is a spasmodic contraction of the cardia, by which not only solids but also liquids are somewhat delayed in their passage into the stomach. This contraction may be increased, both when the cardia is the seat of an organic disease or as a condition accompanying hysteria or neurasthenia. As a result of the cardiospasm, a diffuse dilatation of the œsophagus is often produced. The etiology of cardiospasm and diffuse dilatation of the œsophagus has been a matter of dispute. According to Mikulicz and Meltzer, it is due to a failure of the central inhibitory influences controlling contraction of the cardia. Rosenheim believes that the predisposition to idiopathic dilatation is due to a weakness of the muscular coats; while Krause views it as a functional disturbance of the innervation of the œsophagus causing cardiospasm as well as a weakness of the muscular wall (Tyson). Kronecker and Openkowski succeeded in isolating branches of the vagus controlling the contractility of the cardia. "The contractile force is due to the influence of the central nervous system, as well as the musculature of the cardia itself. The inhibitory influences come through the vagus distribution, and when they are increased the contraction of the orifice occurs" (Tyson).

Etiology.—Cardiospasm is found at times accompanying hysteria and neurasthenia. It occurs after swallowing food that is hard and not sufficiently masticated, or after swallowing food too rapidly or too highly seasoned. Excitement is occasionally a causative factor. It occurs at times after habitual air swallowing. If the air collects, it expands and produces a dilatation of the stomach, and with it a spasm of the cardia, often complicated at the same time with pylorospasm. It occurs at any age, most frequently in females. In this series of 1592 cases of gastric neuroses, there were 25 cases of cardiospasm (1.5 per cent.), 17 being females and 8 males. As to age, 6 were in the second decade, 9 in the third, 7 in the fourth, 2 in the fifth, and 1 in the sixth.

Symptoms.—Cardiospasm occurs in two forms, as the acute or chronic variety. The acute form is only of a few days' duration, the onset being sudden. The attack is accompanied by a burning sensation, with pressure or pain behind the sternum, and dysphagia; as the food accumulates in the œsophagus an effort must be made to force it into the stomach; when this is impossible, the food is regurgitated, causing at times some dyspnœa. At other times it is impossible to swallow food. Regurgitation of the food causes relief of all symptoms. This condition comes on periodically, and during the interval the patient is free from discomfort. In the chronic form there is also a marked dysphagia. Foods are forced into the stomach by the patient with great effort, liquids and semisolids being most easily swallowed; the solid foods must be thoroughly masticated, and are often swallowed by taking small quantities of water at the same time. After a time a diffuse dilatation of the œsophagus takes place, which may hold several hundred

cubic centimeters of fluid. At times less difficulty occurs in swallowing, and occasionally the patient can take a solid meal; however, after the diffuse dilatation of the œsophagus has taken place, the dysphagia becomes constant. If the air swallowed during a meal cannot be eructated, the stomach becomes distended, and on account of the cardiospasm it is impossible for vomiting to take place. The pain and pressure behind the sternum is a common sign, the patient complaining of a sensation as though food were lodged at this point. The dilated lower part of the œsophagus retains the food and regurgitates it after a certain length of time. The contents contain no free hydrochloric acid, pointing to the fact that it did not take its origin from the stomach. The second deglutition murmur is absent. Examination with the bougie reveals important conditions; on introducing it one feels a resistance, which is felt more plainly with the large than with the small-sized bougies; after gentle pressure the resistance gives way and the bougie passes into the stomach. The presence of a diffuse dilatation of the œsophagus above the position of the cardiospasm is demonstrated as follows: A tube is passed in the œsophagus to a point at which food mixed with mucus is withdrawn through the tube. A second tube is now passed into the œsophagus beside the first, but is forced through the resistance; through this tube gastric contents are obtained; if eosin-stained water is introduced through the first tube and plain water through the second, it can be demonstrated that there is no communication between the tubes, as is observed by the return of the fluids separately and not mixed (Rumpel's double tube test). Owing to the lack of nourishment, the general health of the patient fails and there is a gradual loss of weight and strength.

Diagnosis.—In the acute form this is made by the variations in the degree of dysphagia, by the introduction of an œsophageal bougie without difficulty, by the resistance being less from a bougie of large caliber than from a small one, by the absence of the second deglutition murmur, and by the inability of the patient to vomit. In the chronic form the dysphagia lasts over a longer period of time, while symptoms of dilatation of the œsophagus become evident. The condition is recognized by the test of Rumpel, already described. In addition, the examination by means of the bougie reveals a condition similar to that found in the acute form. In those cases accompanied by dilatation, a diagnosis can be arrived at by filling the diverticulum with bismuth and applying the *x*-rays. According to Rosenheim, cardiospasm can be distinguished from other œsophageal disease by means of the œsophagoscope. In this condition there is usually "a characteristic picture": "sharply rising folds of mucous membrane, which converge toward a point in the middle of the lumen and form a rosette-like, more or less rigid closure. The lumen, if it can be recognized at all, is narrow. The mucous membrane of the contracted portion of the œsophagus shows abnormal intense reddening." Cardiac carcinoma is differentiated from cardiospasm from the fact that it occurs in advanced age, that often traces of blood and pus, together with carcinomatous particles, are obtained by the bougie, and that small calibered bougies pass more easily than larger ones. In cardiac cancer there is usually an absence of free hydrochloric acid in the gastric secretion, while in cardiospasm there is either a normal or a hyperacid secretion.

Treatment.—In the acute forms special attention should be paid to the nervous system and the general nutrition of the patient looked into. In order to overcome the cardiospasm large bougies should be introduced into

the stomach and allowed to remain for a considerable length of time. Internally, the bromides, morphine, and cocaine are used with a variable degree of success. Rosenheim recommends the injection at the seat of the affection of a 3 to 5 per cent. eucaine solution, at first twice daily, then less frequently. In other cases he advises cauterization of the inflamed mucous membrane with a $\frac{1}{3}$ per cent. solution of silver nitrate. G. J. Preston relieved the condition by means of hypnotic suggestion in a negro girl, aged eight years, who had not swallowed any nourishment for ten days. In the treatment of the chronic form great care should be exercised not to irritate the œsophagus; for this reason only liquid or semisolid food should be taken; in those cases in which the contraction of the cardia is very great, food must be administered by feeding through a tube; before proceeding with the feeding, lavage of the œsophagus is first practised with the same tube, in order to rid it of mucous masses, food remains, etc. Large bougies should also be introduced and allowed to remain for a considerable length of time. In order to produce a dilatation of the contracted œsophagus and cardia, Schreiber's dilator, Sippy's dilator (rubber bag), and Lockwood's pneumatic bag have been used. When tubal feeding cannot be practised, gastrostomy may be performed. In the cases of Bruning¹ and Erdmann² an opening was made into the stomach and the cardia stretched, in the one case by means of Mikulicz forceps introduced through the opening in the stomach, in the other by the finger of the operator introduced in the same way. The patients made an excellent recovery in both cases.

PYLOROSPASM.

By pylorospasm is meant a spasmodic contraction of the pylorus not due to organic disease. It is usually due to some severe irritation of the mucosa of the pylorus, such as that produced by the acid in superacidity or supersecretion; or it may be due to an ulcer, erosions, fissures, or cancer at the pylorus, while there are other forms entirely of nervous origin. Cases of the latter form have been described in infants by Pfaundler³ and Heubner,⁴ and by Schnitzler,⁵ Brunner,⁶ and Bentejac⁷ in adults. The nervous spasmodic contraction was noted at operation by the last three authorities. In the case of a female, aged twenty-three years, observed by the writer, in whom all the symptoms of pylorospasm were manifested and who was operated on by Finney, no anatomical changes were observed, but entire relief was afforded by pyloroplasty.

Etiology.—In the series of gastric neuroses only 4 cases could be definitely classified as primary pylorospasm (0.3 per cent.). Three of these were observed in females and 1 in a male.

Symptoms.—At the height of digestion, the pylorus suddenly becomes spasmodically contracted, with intense pain in its region, together with eruc-

¹ *Beiträge z. klin. Chirurgie von Bruns*, xlviii, Nr. 2.

² *Annals of Surgery*, February, 1906.

³ *Wiener klin. Wochenschrift*, 1895, Nr. 45.

⁴ *Deut. med. Wochenschrift*, 1901, Versinsbeilage, Nr. 13.

⁵ *Wiener. med. Wochenschrift*, 1898, Nr. 51.

⁶ *Beiträge zur klin. Chirurgie*, 1901, Bd. xxix, S. 520.

⁷ *Thèse de Paris*, 1888.

tations and often vomiting. At first the spasm appears periodically, but as the disorder progresses it may become continuous and produce a spastic contraction of the pylorus, with tendency to dilatation and retention of food. The food is vomited under these conditions, and possesses all the characteristics of that observed in a dilated stomach. Relief is afforded by the vomiting for a period of time varying from one to three or four days, when the same disturbance is again produced. The symptoms found in dilatation are then observed—emaciation, intense thirst, and constipation. The pyloric area, as a rule, is tender to pressure, and contraction may take place near the pylorus, producing a firm mass on palpation, which may gradually disappear. The secretion of acid may be normal or increased, but becomes normal when the motor function is again restored. Chronic gastrosuccorrhœa is frequently observed in these cases and occasionally tetany.

Diagnosis.—This is made by the exclusion of all organic disease and the appearance of the spasmodic attacks of pain in the region of the pylorus, with the appearance of intermittent stagnation.

Treatment.—This consists primarily in properly looking after the underlying neurasthenia. For this purpose change of scene, massage, and electricity, or a rest cure, may be found advisable. The diet should be carefully regulated, all irritating foods being avoided, and the meals should be small, easily digested, and given at regular intervals. The administration of olive oil has been found of great service in pylorospasm, whether due to organic disease or not. When retention is present, gastric lavage with plain water or nitrate of soda solution must be given.

For the pain codeine combined with belladonna is serviceable; hot applications to the abdomen and a thorough lavage usually afford most relief from the pain. If after faithful trial no relief can be obtained from these measures described, the writer has observed excellent results from pyloroplasty. Some authorities recommend a gastro-enterostomy.

PNEUMATOSIS.

By pneumatosis is meant that condition in which the stomach is distended with air and in which expulsion of the same is impossible, causing unpleasant expansion of the stomach and dyspnœa (asthma dyspepticum, Henoch). In a large proportion of cases this condition is partly due to a simultaneous cardiospasm and pylorospasm. Relief is at once afforded when the spasm ceases and the air escapes. Pneumatosis is found secondary to certain gastric disturbances, such as atony and dilatation, or as a primary neurosis accompanying neurasthenia and hysteria. It occurs as a continuous or periodic affection, and appears after nervous anxieties or mental strains. The following case was observed by my colleague Alexis McGlannan: The patient, an extremely neurotic man, was operated on for varicocele. The wound healed by first intention, the sutures and all dressings being removed on the tenth day. On the fourteenth day he complained of abdominal distention, and was observed to be swallowing air. A few hours later he showed signs of acute dilatation of the stomach, collapse, dyspnœa, epigastric distention, etc. His symptoms were promptly relieved by lavage.

Etiology.—Pneumatosis is found more often in males. In this series it occurred in 8 cases (0.5 per cent.); of these, 6 were males and 2 females.

Symptoms.—Pneumatosis occurs in one of two types: in the first type it appears in an acute form intermittently with alarming symptoms causing dyspnoea, collapse, rapid and irregular pulse, and cyanosis. The region of the stomach is greatly distended, often balloon-shaped, and is tympanitic on percussion. The patient is unable to relieve himself by eructating. In the second type the attacks either come on immediately after meals or some time later, or after some exertion; the symptoms are much like those of the acute form, but may become alarming. The gastric contents usually present normal relations in this disorder; occasionally there is a hyperchlorhydria, and again at times hypochlorhydria.

Diagnosis.—This is ordinarily made by observing the symptoms described; all organic conditions, such as dilatation, or atony must be excluded. It is sometimes mistaken for angina pectoris, but in angina there is no continuous distention of the stomach and the expulsion of the gas does not afford relief.

Treatment.—Special attention should be paid to the nervous system. Change of scene, hydrotherapy, and massage will often do much to restore the patient to health. Of the medicines used to give relief, the most important are the bromides, cocaine, or morphine. Boas advises the use of the extract of physostigmine, grain $\frac{1}{4}$ (gm. 0.01), combined with the extract of nuxvomica, grain $\frac{1}{2}$ (gm. 0.02), three times daily. In acute attacks the introduction of the stomach tube will afford instant relief.

PERISTALTIC UNREST (TORMINA VENTRICULI NERVOSA).

Peristaltic unrest is characterized by an excessive and visible peristalsis of the stomach. This condition was first described by Kussmaul as occurring in neurasthenic individuals, in whom excessive peristalsis caused annoying sensations and unpleasant contractions of the stomach. In the cases described by Kussmaul, the peristaltic movements were visible, as a gastropnoia was present at the same time. Peristaltic movements usually take their origin at the fundus of the stomach and run toward the pylorus; the wave, however, may be antiperistaltic also, and run in the opposite direction; occasionally antiperistaltic movements exist alone; cases of this character have been described by Schütz, Cohn, Glax, and recently by Jonas; cases have also been observed occasionally in which peristaltic movements of the intestines were present at the same time.

Etiology.—Peristaltic unrest may occur in one of two conditions: first, due to an obstruction of the pylorus, the stomach exerting itself to overcome the difficulty produced in emptying its contents into the intestines; second, the unrest may occur as a pure neurosis, accompanying neurasthenia or hysteria; as a neurosis it is an exceedingly rare affection, but one case occurring in the series (0.06 per cent.). This was in a man, aged thirty-one years, who complained of movements passing over his stomach some time after meals; the condition was perfectly visible, and was present after almost every meal. Upon careful examination the motor function of the stomach was found increased, while the secretory function was normal.

Symptoms.—The important symptom is the peristaltic movement of the stomach running from the fundus to the pylorus and at times accompanied by antiperistaltic movements. These movements are usually visible, and

are felt by the patient. When the stomach is in its normal position, the patient may be conscious of the movement, but it cannot be seen. Peristaltic movements of the intestines may occur at the same time. These may be observed by means of the x -rays, after giving the patient milk containing bismuth. Jonas observed the antiperistaltic movements in his cases by this method. In addition, other symptoms are often present, such as eructations, nausea, anorexia, and the too rapid evacuation of the stomach (hypermotility). General symptoms of neurasthenia are ordinarily present, such as depression, lassitude, and loss of energy.

Diagnosis.—This is simple if organic obstruction of the pylorus can be excluded, which is usually readily done.

Treatment.—This should be directed mainly toward the neurasthenic condition by change of scene, massage, and hydrotherapy. At times, when the patient is anemic, the best results have followed a well-regulated rest cure. Kussmaul recommends internal and external application of the faradic current. Good results have been obtained by the administration of the bromides, codeine, and belladonna.

SUBACIDITY OR HYPOCHLORHYDRIA.

By subacidity or hypochlorhydria is meant that form of neurosis in which the amount of hydrochloric acid as well as the other constituents of the gastric secretion is lessened. While there are still small quantities of hydrochloric acid secreted in this condition, the quantity is so small that it at once combines with the protein foods. Subacidity is present in certain organic diseases of the stomach, as chronic gastritis and cancer, also during the height of acute febrile diseases. The term nervous subacidity, however, can only be applied to those conditions in which all signs of organic disease of the stomach can be excluded. The condition is frequently found accompanying neurasthenia or hysteria, and is present also in certain lesions of the spinal cord, as *tabes dorsalis*.

In this series 1592 cases, 80 (5 per cent.) represent the number of cases of subacidity or hypochlorhydria; of these, 34 were males and 46 females. As to age, 10 were below twenty, 16 in the third decade, 16 in the fourth, 19 in the fifth, 11 in the sixth, and 8 in the seventh.

Symptoms.—Many cases run their course for a considerable period of time without symptoms; this is especially true as long as the motor function of the stomach is normal, but as soon as this is impaired fermentation begins in the intestines and distention from gas takes place. The symptoms of this condition, however, are not distinctive, and a diagnosis cannot be made until the gastric secretion is examined. Von Noorden¹ first pointed out the fact that the intestines may vicariously take up the work of the stomach in the digestion of food, and that as long as the intestinal digestion is normal, symptoms of gastric disturbances are absent and the patient can maintain his normal weight and strength; as soon, however, as the intestinal functions become disordered by inflammation, diarrhoea sets in and the patient becomes weakened and emaciated.

The following case illustrates this condition: S. M., aged thirty years, student, has been under observation for a number of years (1895 to 1899).

¹ *Zeit. f. klin. Med.*, vol. xvii.

He complained of no gastric disturbance, but permitted himself to be utilized constantly for experimental purposes. His gastric contents showed on eighteen examinations a total acidity varying between 20 and 25, with free hydrochloric acid from 0.0 to 0.07 per cent. On July 3, 1898, after a slight indiscretion in diet, he was attacked with diarrhœa, having from one to four stools a day for a period of two weeks. Notwithstanding a most careful diet, he lost twenty-five pounds in weight. During this period of diarrhœa his stools constantly showed large quantities of undigested food particles. The patient became greatly weakened during the attack, and it took five months for him to again recover his former strength.

A symptom present in 7 of the cases of subacidity was burning with pain about two hours after meals, relieved by alkalies or food and presenting symptoms of acidity. As has been shown by Stockton¹ and Steele² the symptoms must be due to some factor other than the acidity, since this condition does not exist in these cases, and is most likely explained by the existence of a hyperæsthesia of the gastric mucosa.

Diagnosis.—This is arrived at by examination of the gastric mucosa contents and finding a diminution of hydrochloric acid at the same time hysterical or neurasthenic symptoms must be present. By continually observing cases of nervous subacidity one is frequently surprised by the various changes these conditions undergo, often to a normal acidity or even to hyperacidity. Thus a patient aged twenty-four years, male, who was under observation for eighteen months, presented symptoms of subacidity (total acidity from 20 to 25 and free hydrochloric from 0.007 to 0.009 per cent.) on nine examinations; on the tenth examination the acidity was 39 and the free hydrochloric acid 0.1 per cent.; on the eleventh and subsequent examinations the acidity reached 64 and the free hydrochloric acid 0.21 per cent. Hemmeter has termed this condition heterochylia.

Treatment.—The diet should consist largely of carbohydrates. Meat should be given, however, in the most digestible form and finely divided; the food should be given in small quantities at frequent intervals. Much attention should be paid to the general health of the patient, and special treatment should be given to the nervous system. Hydrochloric acid administered in 10 to 20 drop (1 cc.) doses after meals is a useful adjuvant. Lavage of the stomach is rarely necessary unless the motor function is markedly disturbed and at the same time leads to fermentation. The use of massage and electricity (intragastic) is also serviceable in many cases. The saline waters, such as Kissingen or Wiesbaden, are useful in cases of subacidity so long as the motor function is normal; so soon as this is disturbed their use should be discontinued.

ANACIDITY OR ACHYLIA GASTRICA.

The term achylia gastrica was first introduced by Einhorn³ to indicate a condition in which there is an absence of gastric secretion. The term achylia gastrica is simply meant to designate a symptom, the condition underlying the same being a severe form of chronic gastritis or atrophy of

¹ *Journal of the American Medical Association*, January 11, 1903.

² *Therapeutic Gazette*, January 15, 1906.

³ *New York Medical Record*, June 11, 1892.

the mucous membrane or some condition wholly neurotic in origin. It is not our province to discuss here the former class of cases, those which are accompanied by a complete atrophy of the gastric mucous membrane. Fenwick¹ first described this condition in certain cases of pernicious anæmia. The use of the term achylia gastrica is best restricted to those classes for which Einhorn first adopted it. These are those cases in which there is a complete lack of gastric secretion which persists for many years without ending fatally, and in which the general condition of health frequently improves. The patient suffering with this condition often presents no subjective symptoms whatever, and can take many forms of food without the slightest discomfort, the small intestine vicariously assuming the function of the stomach.

To this class of cases belong those described by Einhorn,² Ewald,³ A. Jones,⁴ Boas,⁵ Rosenheim,⁶ Martius and Lubarsch.⁷ In this connection Einhorn describes a patient kept under observation for four years and who improved in health during this time, while in another the condition persisted for forty years. Ewald reports a case in which the absence of gastric secretion persisted for two and one-half years and, nevertheless, the patient gained forty-two pounds in weight. In a case of the writer's this condition had persisted for twelve years, with gain in weight and with but few attacks of intestinal disturbance.

Etiology.—From the more recent investigations it seems doubtful, in most instances, whether a complete absence of gastric secretion is due to a neurasthenic condition alone, because, while the neurasthenic condition may improve and disappear, the gastric secretion remains absent permanently.

According to many writers it is probable that a more or less pronounced chronic gastritis is present in a large proportion of cases of achylia gastrica. According to Martius, achylia gastrica is produced by two conditions: First, simple achylia gastrica, a primary secretory debility of the stomach that may be either congenital or developed early from hereditary predisposition; and second, atrophy of the gastric mucosa producing a secondary achylia gastrica. Simple achylia gastrica is found in neurasthenic patients, and is associated with inherited weakness of the nervous system. It may remain latent for years, as has been already stated, without making serious inroads upon the general health. This is especially observed in those patients in whom the motor function of the stomach remains normal and the intestinal functions are undisturbed. There are transitions from the simple forms of achylia gastrica in which there are none or but the slightest changes in the mucous membrane to that form ending in complete gastric atrophy.

According to Stockton, the belief that achylia gastrica may in some instances begin as a simple depression of the functional activity of the secretory glands of the stomach, the result in some cases of neurasthenia, in others as reflex irritation, appears to be gaining rather than losing ground. According to this observer, with the improvement in health gastric secretion is increased,

¹ *Lancet*, July, 1877.

² *New Yorker Medicine Press*, September 4, 1888.

³ Under the head of *Anadenia Ventriculi*, *Berlin. klin. Wochenschrift*, 1892, Nrs. 20 and 27.

⁴ *New York Medical Journal*, May 27, 1893.

⁵ *Münch. med. Wochenschrift*, 1887, Nrs. 21 und 42.

⁶ *Berlin. klin. Wochenschrift*, 1888, Nrs. 51 und 52.

⁷ *Achylia Gastrica*, Leipzig and Vienna, 1897.

and disappears again with nervous strain. When it is absent for any length of time, it is probable that considerable atrophy of the mucous membrane has appeared, and yet in certain cases Stockton has observed the secretion appearing after it has been absent for a period of a year or more. Billings¹ reports four cases to show that achylia may exist in persons who have no organic disorder of the stomach, and that it may therefore be caused by a general neurotic condition. On the other hand, from an observation of some patients for at least fifteen years, Stockton² believes that, in the absence of or with a very low standard of gastric secretion, individuals are always found to have impaired health, even with relatively good intestinal digestion. He therefore considers the question of stomachic digestion of the greatest importance.

Achylia gastrica is usually found after the thirtieth year of age; it is rarely found in younger persons, Litten and Rosengart,³ however, report cases occurring at the age of eighteen years, and Einhorn⁴ at the age of twenty-five years. There is apparently no difference in the frequency of occurrence in males or females. In this series the number of cases of achylia gastrica is 114; of these, 2 represent that form known as atrophy of the stomach, and cannot be considered here, leaving 112 cases (7 per cent.) in all, 57 being females and 55 males. As to age, 2 were below twenty, 13 in the third decade, 19 in the fourth, 33 in the fifth, 35 in the sixth, and 10 in the seventh.

According to Leipschutz,⁵ the gastric secretion has a general tendency to decrease in individuals over fifty years of age, and by his statistics, the percentage of cases of achylia in persons over fifty years of age was 37 per cent.; according to this series, the percentage is 40.

Pathology.—There have been so few cases of achylia gastrica in which autopsies have been performed, that it is not known whether lesions occur in all cases. According to Martius and Lubarsch, and Strauss,⁶ a more or less marked gastritis exists in a large proportion of cases of achylia. On the other hand, there is no means of determining whether the gastritis is the cause or the result of the achylia. Einhorn⁷ maintains that there are many cases of achylia in which the mucous membrane of the stomach presents no pathological change whatever, which accounts for the fact that a repair of the condition is sometimes found. Small fragments of the mucous membrane that are broken off by the stomach tube usually present some degenerative change. We are not justified, however, in drawing conclusions from the examination of small bits of mucous membrane. This fact has been emphasized by Lubarsch and Leuk, who state that but little reliance can be placed upon these examinations, as we cannot draw general conclusions concerning the entire stomach from only small fragments of mucosa. The mucous membrane in these cases of achylia gastrica is very vulnerable, and it is not an infrequent occurrence that bits of mucosa are torn off. The glands show a condition of granular degeneration with considerable round-cell infiltration presenting the picture of a granular gastritis. It is generally believed by most authorities that notwithstanding the impossibility of judging the general condition of the mucous membrane from fragments

¹ *Virginia Medical Monthly*, June 21, 1907.

² *New York Medical Journal*, February 17, 1906.

³ *Zeit. f. klin. Med.*, vol. xiv.

⁵ *Ibid.*, vol. xii, p. 426.

⁷ *New York Medical Record*, July 6, 1895.

⁴ *Archiv f. Verdauungsk.*, vol. i, No. 2.

⁶ *Virchow's Archiv*, Bd. cliv.

of a small area, in the largest proportion of cases of achylia gastrica there is some degeneration in the mucous membrane of the stomach, and there is no great amount of evidence at present to indicate that this condition can occur without some anatomical change in the stomach.

Symptoms.—In regard to the symptomatology, Einhorn¹ divides achylia gastrica into three groups: (1) In individuals having no gastro-intestinal symptoms whatever, and who are in good general health. (2) Patients presenting a greater or less number of gastric symptoms. (3) Patients having apparently no gastric symptoms, but presenting marked intestinal disturbances. The smallest number of cases are contained in the first group, in which all subjective symptoms are absent. The individual presents no loss of weight, the appetite remains unimpaired, and the diseased condition is usually noted by accident. In this series of 112 cases, only 8 can be counted in this group. The following case illustrates this condition: J. M., male, aged thirty years, presented himself for treatment for a neuralgic headache. He had always been in good health and had never had any serious diseases. On examination he was found to be a very robust-looking man with normal organs; by accident a test breakfast revealed a most interesting condition; only 20 cc. of gastric contents were obtained, with a total acidity of 2; there was no hydrochloric acid, pepsin, or rennin; the erythrodextrin reaction was absent. This condition persisted even although there were no symptoms of any gastric disturbance. Twenty examinations of the gastric contents were made at varying intervals, always with the same results. This demonstrates how an individual may continue to maintain his strength and weight, even with an entire cessation of the gastric secretion.

The largest proportion of cases is found in the second group, in which the symptoms are mainly gastric; in this series of 112 cases, 69 belong to this group. The symptoms are loss of appetite, sensations of pain and pressure in the region of the stomach, and occasionally vomiting. The pains are generally mild, however, often merely a sensation of burning and pressure occurring soon after meals and persisting for some time; in some cases there may be no pains whatever, while in others the pain may occur with great severity. Nausea and vomiting occur rarely, in but 17 cases of this series. The vomited matter usually contains undigested food particles. Eructations and pyrosis are observed at times; occasionally symptoms are presented which are similar to those found in hyperchlorhydria; that is, the appearance of pain one or two hours after meals, relieved by the ingestion of food. This symptom was noted in 11 cases in this series. Nervous symptoms are also frequently present, such as headaches, vertigo, depression, and impaired sleep. The following case illustrates the condition of achylia gastrica in which symptoms of hyperchlorhydria were constantly present. S. R., female, aged fifty-three years, had suffered from gastric trouble for one year. There was a burning sensation in the stomach, heartburn, and pain from one to two hours after meals. These pains could be relieved by drinking a glass of milk, but became unbearable if relief was not promptly given. The patient also complained of occasional nausea, headaches, and great nervousness. On examination she was found to be robust, with no loss of weight. The abdomen was normal; on inflation the stomach extended to one finger's breadth above the umbilicus. The gastric contents obtained after an Ewald

¹ *Diseases of the Stomach*, 4th edition, p. 380.

test breakfast were small in quantity, 25 cc., with a total acidity of 4; free hydrochloric acid and rennin were absent; the erythrodextrin reaction was absent. Similar results were obtained in six other examinations. The symptoms of hyperchlorhydria in these cases is explained as due to a hyperaesthesia of the gastric mucosa.

The third group contains those patients having apparently no gastric disturbance, but presenting intestinal disturbances. In the series of 112 cases these number 35. The most prominent symptom is diarrhoea. The patient complains of little or no disturbance of the stomach, but is affected with symptoms that are apparently referable to some marked disturbance of the intestines, complaining of gurgling in the bowels and intestinal colic. Frequently diarrhoea alternates with constipation. The diarrhoea is probably occasioned by the passage of undigested food from the stomach into the intestines, mechanically stimulating peristalsis. In addition, as Leipschutz¹ points out, a large number of bacteria pass through the stomach, the disinfecting power of the gastric secretion being absent, and thus cause decomposition in the bowels. The following case illustrates this form: J. W., aged fifty-nine years, complained of diarrhoea which had troubled him for eight months. Occasionally he had periods of relief, during which there was a marked constipation. These periods, however, only lasted from two to three days, and were immediately followed by attacks of diarrhoea. The patient had lost but little weight, but the persistent diarrhoea occasioned considerable exhaustion. On examination the patient was found to be a very robust man, whose organs appeared to be normal; the stomach on distention reached to the umbilicus; the gastric contents gave an absence of free hydrochloric acid, with a total acidity of 4. Pepsin, rennin, and the erythrodextrin reactions were absent. Eight other examinations showed the same condition. This case is interesting, proving that with a total absence of gastric secretion an entire absence of gastric symptoms may exist, and that the symptoms may be referred almost entirely to the intestines.

Examination of the gastric contents is of the greatest importance in the diagnosis of achylia gastrica. The following are the characteristics of the gastric contents. The amount is very small, containing but a small quantity of fluid, with bits of unaltered bread; in the morning before the ingestion of food the stomach is entirely empty, with no traces of food from the previous day. The gastric contents are neutral or slightly acid, the total acidity varying between 2 and 6. Hydrochloric acid is not present, pepsin and rennin are entirely absent, although the rennet zymogen may still be present; the test for propeptones and peptone reveals negative results, and there is an absence of mucus. Fragments of mucous membrane often appear in the contents, indicating the great vulnerability of the mucous membrane. The motor function of the stomach in achylia is usually normal.

Diagnosis.—For the most part this is not difficult, the examination of the gastric contents leading to a correct diagnosis; repeated examinations are, however, necessary to confirm it. The most important signs are an entire and constant absence of hydrochloric acid, an almost entire absence of the ferments, and a very low total acidity. As in nearly all of the cases some alteration has taken place in the gastric mucosa, whether or not the achylia is the cause or effect of the change, it is difficult in many instances

¹ *Archiv f. Verdauungsk.*, vol. xii, p. 436

to distinguish this condition from a gastric gastritis. One must rely mainly upon the presence of the nervous symptoms and the complete absence of mucus; a fluctuation in the gastric secretion will occasionally indicate the nervous origin of the disorder. This condition is distinguished from carcinoma by the absence of the typical signs of cancer. The degree of acidity is much higher in carcinoma even when free hydrochloric acid is absent; lactic acid is usually present as well as the *Oppler Boas bacillus*; the latter is not found in achylia. There is a constant finding of occult blood in carcinoma never observed in achylia; in 50 examinations from 24 cases of achylia in this series, occult blood was never found in the feces at any time. Hemorrhages, also, are never observed in achylia. When cancer is advanced, no difficulty is present in the diagnosis.

Course and Prognosis.—Patients suffering from achylia gastrica may be affected for many years without having serious inroads made in their general health. They may even gain flesh when the course is latent for a long period of time. As long as the motor functions of the stomach remain normal and the intestine takes up the functions of the stomach vicariously, no serious results are produced in the general nutrition; when, however, one or other of the functions are disturbed, the symptoms of achylia begin to manifest themselves and the general nutrition becomes markedly disturbed. Since a chronic gastritis is present in most instances, an absolute cure is impossible, even though the symptoms are entirely relieved.

Treatment.—This is mainly dietetic, and although it is necessary to restrict the diet materially in this condition, it is most important to insist on the ingestion of sufficient food, as many of these patients are weak and have lost flesh. The question as to quantity of food taken, therefore, should be carefully considered with an attempt to increase the general nutrition as well as the body weight. In accomplishing this object these facts should always be held in mind, namely, to maintain the motor function of the stomach in its normal condition, as well as to prevent any disturbance of the intestinal canal, since the intestine acts vicariously and digests the food for the stomach. It is important to arrange the diet so that it can be acted upon easily by the intestinal juices. The food must be broken up into as fine particles as possible and should to a large extent be given in liquid and semiliquid form. Of the liquids, broths, such as barley, rice, or chicken, are to be recommended. Vegetables are usually well taken; cereals should be eaten after the cellulose has been removed. Peas and beans strained and eaten in purée form, as in broth, are especially useful, since they contain quite a large percentage of protein. Potatoes and rice should be eaten cooked with broth or milk, or as a mush. Eggs are best taken soft boiled. Meats must be given in the most digestible forms, *i. e.*, brains, scraped beef, boiled sweetbreads, and these only in small amount; raw oysters and boiled fish are also permissible. Milk is occasionally imperfectly digested in this condition, and cream kefir, koumyss, or matzoon may be substituted for it. Butter may be eaten on crackers, stale bread, or toast. Such beverages as tea, coffee, cocoa, and small quantities of wine may be allowed. In those cases in which symptoms of hyperchlorhydria are manifested, the taking of liquids from one to two hours after meals (such as milk, albumin, or even water) will afford relief.

OUTLINE OF A DIET FOR ACHYLIA GASTRICA.

		Calories.
8 A.M.	200 gm. milk flavored with tea	135
	60 gm. stale bread (154) with 40 gm. butter (326)	480
	2 soft boiled eggs	160
10 A.M.	100 gm. scraped beef (119) with	
	60 gm. stale bread or toast (154) or	
	100 gm. stewed chicken, minced (106)	273
12 M.	Bouillon with egg	84
	100 gm. of fish boiled and minced	80
	100 gm. mashed and baked potatoes	127
	100 gm. spinach	166
	60 gm. stale bread (154) with 40 gm. butter (326)	480
4 P.M.	200 gm. cocoa with milk	135
6 P.M.	200 gm. milk flavored with tea	135
	200 gm. milk and rice	353
	50 gm. of bread and 30 gm. butter	343
10 P.M.	100 gm. of milk (kefir or koumyss)	67
		<hr/> 3018

Lavage and intragastric faradization may be resorted to in those instances in which the gastric motility is somewhat impaired. Drugs are not always required in achylia gastrica. In some cases dilute hydrochloric acid may be given, well diluted in 15 drop (1 cc.) doses, three or four times at intervals of fifteen minutes after meals with benefit. For the anorexia, which may be present, strychnine combined with the bitter tonics is recommended. In those instances in which the general nutrition has become very much depressed, and in which diarrhœa is or is not present, excellent results may be obtained by a systematic rest cure treatment.

ACORIA.

By acoria is meant that condition in which there is an absence of the sensation of satiety. The patient does not feel that he has eaten enough, no matter whether he partakes of a large or a small meal; the appetite is not increased; it may even be diminished. The cause of this disorder has not been determined; it has been stated that acoria is due to an overstimulation of the hunger centre, but as relief of hunger does not relieve the sensation of the absence of satiation, this theory is untenable. It is also held that this condition is due to loss of sensation in the stomach itself.

Etiology.—Acoria occurs as a primary disorder associated with neurasthenia or hysteria, or is observed secondary to other diseases, such as diabetes or disorders of the male sexual organs. Sorrow, sudden shock, or excitement are among the etiological factors concerned in its production. In this series of 1592 cases it occurred five times (0.3 per cent.); of the 5 cases, 3 occurred in males and 2 in females. Two males and two females were between thirty and forty, and one male between forty and fifty years of age.

Symptoms.—The affection is characterized by an absence of the sensation of satiation, no matter how large the meal may be. In consequence of the inordinate eating, atony and gastritis may develop. Acoria may at times be transformed into bulimia, which Boas explains by the fact "that hyperæsthesia of the gastric nerves may be followed by anæsthesia as a

reaction." The gastric secretion is usually normal; in this series normal acidity existed in 4 and subacidity in 1; the gastric motility was normal.

Diagnosis.—Acoria must be differentiated from polyphagia and bulimia; in acoria there is an entire absence of the sensation of satiation, while in polyphagia the feeling of satisfaction is delayed to such an extent as to create a constant desire for more food. In bulimia there is an abnormal desire for food, but this desire can be satisfied.

Treatment.—This must be directed toward the cause, which in most instances involves treatment of the general neurasthenia by means of rest, change of scene, hydrotherapy, massage, and electricity. Entire relief was affected in two cases by a rest cure combined with psychotherapy. The meals should be carefully regulated, the patient should be required to eat slowly and masticate thoroughly. Strychnine in ascending doses has been used with some success.

ANOREXIA NERVOSA.

Anorexia is characterized by a marked diminution in or the absence of the sensation of hunger, combined with a complete loss of appetite. Penzoldt points out that the terms hunger and appetite should be separated, as they are not synonymous; according to this author, hunger is defined as the warning for food, while appetite is the pleasure in partaking of it. Anorexia may accompany various organic or functional diseases, or may be a primary neurosis, accompanying neurasthenia or hysteria (anorexia nervosa). In this condition the loss of appetite may be so great as to cause a most marked disgust or repugnance for food. The cause of nervous anorexia has been attributed to a depressed state of the hunger centre, while it is generally believed that this affection is produced by a "special hyperæsthesia" of the mucous membrane of the stomach (Rosenthal).

Etiology.—Anorexia may be primary or secondary. It is primary as a manifestation of hysteria and neurasthenia; it is secondary to such diseases as cancer of the stomach, chronic gastritis, gastropotosis, acute febrile conditions, chronic nephritis, and other organic diseases. Anorexia nervosa usually begins after some great mental strain, excitement, or anxiety. It is not uncommonly found in men with sexual disorders (Peyer) and in women affected with uterine disease, and is often observed in a most aggravated degree in the insane (anorexia mentale). Opium habitués are commonly affected with this disorder. It is most frequently observed in females, and especially between the ages of fifteen and forty years. In this series of 1592 cases there were 48 cases of nervous anorexia (3 per cent.), 11 being males and 37 females. As to age, 19 were in the second decade, 12 in the third, 10 in the fourth, 4 in the fifth, and 3 in the sixth.

Symptoms.—After some great mental strain or worry a loss of appetite begins to manifest itself, the digestive powers are still normal, but the patient shows a lack of desire for food. This lack of appetite becomes more and more decided until the stage of absolute repugnance for food is reached, and the patient loses weight and strength, becomes anæmic and pale, the pulse becomes slow and weak, the urine scanty, and marked constipation is evident. With these symptoms, others of a nervous character manifest themselves, and the patient becomes irritable, restless, and sleepless, and, if forced

to take food, vomits. If the condition still progresses, symptoms of a very serious character arise, such as inanition with delirium, slight fever, and exhaustion, and the patient may die. When the disease is well-marked, the patient presents the appearance of having tuberculosis; the skin is dry, the abdomen is markedly retracted, the eyes are sunken, the pupils are dilated, and the mucous membranes are pale. Death may be due to exhaustion, pneumonia, or bacterial infection from loss of resistance.

Diagnosis.—The presence of other nervous manifestations, together with the anorexia and absence of all organic disease, easily leads to a correct diagnosis. In all cases, in arriving at a diagnosis organic diseases accompanied by anorexia must be excluded.

Treatment.—In the milder forms much can be accomplished by insisting that the patient take sufficient nourishment. In order to make this possible the food should be varied and the patient's taste so far as possible be consulted. The bitter tonics at times play an important role; of these, gentian, colomba, quinine, and strychnine are often useful, especially when combined with dilute hydrochloric acid; orexin (5 grains three times daily in capsules) and the bromide of soda have been tried. Lavage with a bitter infusion, or with a 0.5 per cent. solution of sodium chloride, has been recommended by Kussmaul and Fleiner. The nervous system should be built up by means of massage, hydrotherapy, and electricity. In all serious forms the patient should be isolated in a hospital or sanitarium, and placed under a strict rest cure, with forced feeding. The writer feels confident that the lives of a large number of his patients were saved by insisting upon this method of treatment. In those instances in which the patient still refuses to take food, gavage should be practised. When improving, some form of iron, such as the albuminate or Bland's pills, will be found serviceable.

SITOPHOBIA.

The term sitophobia was first introduced by Einhorn to indicate a special neurosis of the stomach, manifested by marked fear of food. He pointed out that if this condition continues the dyspeptic symptoms gradually increase, even after taking the smallest quantities of food. In consequence the patient loses much flesh, becomes anæmic, and develops other neurasthenic symptoms, such as dizziness, headaches, lassitude, etc. The fear is due to the unpleasant sensation and pain which occur after eating, in consequence of which the patient gives up one form of food after another until extremely small quantities of nourishment are taken and signs of inanition are produced.

Etiology.—Sitophobia may exist secondarily to organic or functional disorders of the gastro-intestinal tract, or be a neurosis due to hysteria or neurasthenia. As a secondary condition it is found in ulcer, chronic gastritis, cancer of the stomach, chronic constipation, chronic diarrhoea, gastralgia, and enteralgia. Quoting from Hosslin, Einhorn says: "Such fears are not possible in perfectly normal people; for the expression 'phobia' presupposes the existence of an abnormal fear, based upon false ideas. If this fear is not normal but justifiable—if, for instance, a patient suffering from acute enteritis is afraid to eat cabbage or prunes, or if a person who is afflicted with urticaria after partaking of strawberries or lobster avoids these dishes—then we do not have to deal with so-called phobia, but with a

very sensible train of reasoning; but if this fear is abnormal, some psychic change has taken place. Sitophobia is, therefore, like all other phobias, of cerebral origin." With reference to these remarks of Hosslin, Einhorn replies: "Sitophobia is certainly found in people who do not suffer from any psychic disturbance. The patients are afraid to eat because they have pain afterward. This fear is justifiable; but in order to effect a cure it must be overcome, and, therefore, the treatment of this symptom (sitophobia) plays an important part." In the series of 1592 cases of gastric neuroses there were 24 cases of sitophobia (1.5 per cent.), 18 being females and 6 males. As to age, 4 were in the second decade, 7 in the third, 3 in the fourth, 6 in the fifth, 3 in the sixth, and 1 in the seventh.

Symptoms.—On account of pain or uncomfortable sensations in the stomach the patient gives up one food after another until he subsists on a very small quantity of liquid food; in consequence of which the symptoms of inanition are produced. These are indicated by severe headaches, nervous manifestations (vertigo, general aches and pains), great loss of strength and flesh and marked anæmia. Many of these patients consume only from 400 to 1000 calories of food per day; if this condition persists, the emaciation and loss of flesh continue, until the patient finally succumbs.

Treatment.—It is necessary to insist that the patient make every effort to take his nourishment, even though it produces pain. In following out this plan he will observe that food which at first occasioned uncomfortable sensations gradually loses this effect and can be taken without fear of distress. At first liquid food alone should be given in the form of milk or egg albumen, and gradually the diet should be increased to the soft and then to the solid form. In severe cases marked results are obtained by a well-regulated rest cure. It is an excellent plan to persist with liquid food, such as milk and eggs, for from ten to twelve days of the rest treatment, then increasing to very large quantities, and later suddenly placing the patient upon a generous diet without attracting his attention to the same. The patient gradually gains much in weight and strength by this plan of treatment. Occasionally the bromides, belladonna, and hyoscyamus are useful in reducing the sensitiveness of the stomach.

INCONTINENCE OF THE PYLORUS.

Attention was first called to insufficiency or incontinence of the pylorus by de Sère and later by Epstein. The pylorus is insufficient when, during digestion, it is incontinent and fails to prevent the chyme from proceeding into the intestines at too rapid a rate.

Etiology.—The condition may be due to an organic disorder, or may exist as a neurosis. The organic conditions that may produce the insufficiency are carcinoma or ulcer of the pylorus or chronic gastritis. Boas has observed it in duodenal stenosis in which the pylorus was dilated by the material accumulating above the stenotic area. As a neurosis, incontinence of the pylorus is a very rare affection. In the series studied there were but two cases (0.1 per cent.), one in a male aged twenty-seven years, the other in a female aged forty-one years. The following case represents the condition: J. F., aged twenty-seven years, complained of gastric disturbances for six months. He had frequent eructations and abdominal distention,

together with general neurasthenic symptoms, such as lassitude and dizziness. After numerous attempts to withdraw a test breakfast, no gastric secretion could be obtained, even at intervals of thirty and forty-five minutes, but considerable quantities of biliary fluid were obtained; four attempts at inflation proved fruitless, but produced a marked increase of the intestinal tympany. This patient was greatly relieved by a carefully regulated diet (small meals given at frequent intervals) and the use of internal applications of electricity.

Symptoms.—This condition becomes evident by the fact that on inflation of the stomach this organ does not become filled and distended, but instead a gurgling takes place at the pylorus, the escape of air causing increased intestinal tympany. In addition, the stomach empties itself too rapidly after test meals and other meals, and there is a reflux of considerable quantities of intestinal contents, bile, and pancreatic secretion into the stomach. This is observed in almost every lavage. Because of the very rapid evacuations of the stomach, diarrhœa is a very common symptom.

Treatment.—Because of the passage of the indigested food into the intestines and the diarrhœa, food should be given in a very digestible form and finely minced. Einhorn recommends direct gastrofaradization and occasional lavage of the stomach. In some instances strychnine has been of service.

ATONY.

Atony of the stomach is that condition in which the muscular walls have lost their tonicity, with a resultant motor insufficiency, in consequence of which the stomach is unable to pass its contents into the intestine at the normal rate. Although this condition has long been recognized, great confusion still exists concerning its significance. By some authors it is classified as a purely nervous disorder, by others as a form of gastrectasia, while some pass it by in a cursory manner.

Kussmaul¹ was among the first to make a distinction between atony and dilatation. He showed that in gastrectasia due to stenosis of the pylorus or of the duodenum, vomiting is very frequent during the entire course of the disease, and is a characteristic sign, while in atony it rarely occurs. Oser² attempted to distinguish between dilatation and atony, and stated that in actual dilatation lavage is of great benefit, but that in atony it is of little value. Naunyn³ laid stress on fermentation in dilatation of the stomach, and showed that in muscular insufficiency fermentation is usually produced. Minor forms without fermentation, he believes, should be excluded from the chapter of true gastrectasia. Germain See likewise distinguished clinically between dilatation and atony. For clearer views concerning this subject we are much indebted to Boas,⁴ who described this disorder at length as a separate pathological condition, devoting a special chapter to it, and called it myasthenia (muscle weakness) or muscular insufficiency of the first degree.

Etiology.—As regards causation, atony may be of two kinds, primary and secondary. Primary atony is found in persons who have been in the habit of

¹ *Volkman's klinische Vorträge*, 1880, Nr. 181.

² *Ursachen der Magenerweiterung, Wiener Klinik*, 1881.

³ *Deut. Archiv f. klin. Med.*, Bd. xxxi, 1882, S. 225.

⁴ *Specielle Diagnostik und Therapie der Magenkrankh.*, ii Theil, iv Auflage, S. 89.

consuming large quantities of indigestible food; the excessive use of fluids especially predisposes to this disorder; frequently, however, no such cause is assignable. Atony may be secondary to many other diseases, such as those of the brain and cord, typhoid fever, anæmia, tuberculosis, and diseases of the gastro-intestinal tract, such as gastroparesis, enteroparesis, chronic constipation, chronic gastritis, and nervous dyspepsia. Cholelithiasis is not an infrequent cause (Boas). According to Peyer, it is often found as a neurosis secondary to disease of the generative organs in males. Bamberger finds it frequently congenital, and according to Zweifel it is not uncommon in children, due, as he believes, in many cases to improper feeding. Kundrat also finds it in children, and especially among those that have been subjected to many attacks of gastro-intestinal catarrh. Pfungen has shown that atony of the stomach often originates during the period of puberty; it is not improbable that the precocious appetite of this age, leading to the consumption of much indigestible food, is the cause of the disturbance. For the same reason it is not uncommon after typhoid fever. A very frequent cause is gastro-intestinal auto-intoxication. No age seems to be exempt from the disorder, and it appears with equal frequency in males and females. In the series of 1592 cases there were 147 cases of atony of the stomach (9.25 per cent.), 79 being females and 68 males. As to age, 7 were in the first decade, 26 in the second, 26 in the third, 30 in the fourth, 25 in the fifth, 19 in the sixth, and 14 in the seventh.

Symptoms.—Most patients complain of a loss of appetite, although in rare instances the appetite may be fully maintained, at least in the first stages. When the condition is secondary to nervous disorders the appetite may even be increased (Peyer). A feeling of pressure or fullness comes on, usually during or after meals, and this is especially marked after the ingestion of fluids. In light forms the distress reaches its height immediately after meals, and gradually passes off during the next hour, to be again aggravated by the smallest amount of food. In severer forms it may be so great as to continue with intensity for hours after meals, and become still more aggravated when food is taken again. This feeling of pressure is accompanied by heartburn, pyrosis, and eructations of gas, but rarely by vomiting. The quality as well as the quantity of food ingested is productive of this symptom; fluids are most likely to induce pressure. Constipation is almost a constant feature and headache of a dull character is often present, and may at times lead to actual vertigo. Nervous symptoms of various kinds may be present, such as palpitation of the heart and indefinite pains, and on this account the disorder is frequently mistaken for neurasthenia.

On physical examination the stomach is found to be enlarged, so that the greater curvature reaches to or below the level of the umbilicus. Peristaltic and antiperistaltic movements of the walls of the stomach may occasionally become visible. With but small quantities of fluid (from 250 to 300 cc.) in the stomach, splashing sounds may be readily produced. In atony the stomach is not only enlarged but its motor function is also markedly impaired, and it does not propel its food into the intestine at the normal rate. The propulsive force of the stomach may be measured by the salol test of Ewald and Sievers, but the test dinner of Leube is of greater value. Under normal conditions the stomach will be found empty in from six to seven hours. Should particles of food still be present after this time the motor function of the stomach is much impaired. A most important test is the condition of

the stomach before the ingestion of food (Boas). If the contents of the stomach be expressed in the morning before taking food, the stomach will be found entirely empty and free from all food remains. This is not so in cases of gastrectasia, in which greater or smaller quantities of food will be found. Boas has devised still another test, which has proved of great service. The test supper of Boas consists of white bread with butter, cold meat, and two cups of tea taken at 8 P.M. In atonic conditions the stomach will be empty the next morning, but in gastrectasis it will still contain food remains at that time.

Examination of the gastric contents is of great importance. The expression one hour after an Ewald test breakfast shows large quantities of solid contents, not separating into the characteristic three-layered fluid of gastrectasis and not containing yeast spores or sarcinae. Upon chemical examination the contents show in most cases a normal proportion of hydrochloric acid, pepsin, and casein ferment. According to Boas, at the very beginning of atony of the stomach, through constant mechanical irritation of food upon the walls of the stomach, an increased acid production results; in fact, the irritation may be so great that even hypersecretion may be produced. In other cases there may be a condition of subacidity. The findings in this series were hyperacidity 36, normal acidity 60, subacidity 20, anacidity 14, and alternating acidity in 17.

Among the frequent complications of atony may be mentioned dislocation of the stomach (gastroptosis), of the bowel (enteroptosis), and of the right kidney. Litten endeavored to demonstrate that dilatation of the stomach may cause the liver to move downward, carrying with it the right kidney, but very few accept this explanation and Nothnagel claimed that Litten's cases were for the most part really atony. Although dislocation of the right kidney is quite commonly associated with atony, he maintained that this association was merely a coincidence. It is probable that the atony in these cases is secondary to a gastroptosis. Atony of the intestine is not an infrequent concomitant of atony of the stomach; indeed, both may be present for years, and it may be difficult to tell which is the primary trouble. Gastric vertigo, the vertigo dyspeptica of Trousseau,¹ which this writer believed to be caused by chronic gastritis, probably depends in most cases upon atony (Boas). The writer has reported a case of this kind. According to Boas² the so-called asthma dyspepticum is not uncommonly found in atony of the stomach.

Diagnosis.—Atony must be differentiated from nervous dyspepsia and gastrectasis. According to Bamberger³ the variability and rapid change of symptoms, the presence of other nervous symptoms, the normal and increased appetite, and the absence and rapid disappearance of the gastric disturbance, distinguishes nervous dyspepsia from atony. At times a diagnosis becomes very difficult and sometimes impossible. It must not be forgotten that atony is frequently a complication of nervous disorders.

From gastrectasia, atony is diagnosed by the absence of food remains in the stomach in the morning before taking food; by the absence of the three-layered fluid of gastrectasia, and by the absence of sarcinae and yeast

¹ *Vertigo Dyspeptica*, *Gaz. des Hôpitaux*, 1862.

² *Archiv f. Verdauungsk.*, 1896, Bd. ii, S. 444.

³ *Krankh. der Chylopoet. Systems*, *Virchow's Handbuch*, Bd. vii, I, 1855, p. 270.

spores. There is a marked diminution in the secretion of urine in gastrectasia, but not in atony.

Prognosis.—Atony of the stomach is a chronic disorder and may last for years. It is quite amenable to treatment, and, although it may not be perfectly cured, the patient may be relieved of all suffering. It may pass into gastrectasia after years, but this is a rather rare occurrence.

Treatment.—Since atony is frequently caused by injudicious and too rapid eating, people with feeble digestive powers should exercise special caution to eat slowly, masticate thoroughly, and avoid indigestible food. Persons suffering from atony of the stomach should eat small quantities of food at frequent intervals. Since water is not absorbed in the stomach to any extent, it is advisable that the quantity of fluids taken should not exceed $1\frac{1}{2}$ liters a day; this amount should include all fluids: coffee, tea, soups, etc. If the thirst is very great, enemata of water or nutrient enemata may be administered.

The use of milk in large quantities has been recommended, but is not to be advised when the patient is able to go about, since the weight of large quantities of milk may overdistend the stomach; when, however, a rest cure is instituted, milk is often well borne, even in very large quantities. The diet in atony of the stomach varies according to the nature of the gastric secretion. In cases of superacidity a liberal meat diet, consisting especially of chicken, beef, mutton, or ham, is recommended; fish, eggs, hard and soft boiled, are also permissible; the vegetables should be selected with care; carrots, peas, and cauliflower may be given, but must be mashed and strained so as to rid them of cellulose. Potatoes, rice, and grits are also allowed. Butter is the form of fat best suited to this condition.

Alcoholic stimulants are, as a rule, not well borne, and their use should be prohibited; in a limited number of cases alcohol in the form of a light wine acts as a stomachic and may be prescribed. In those cases in which there is an absence of a diminution of acid in the gastric secretion the lighter forms of meat, such as the white meat of chicken or fish, sweet-breads, stewed chicken, or raw scraped beef, should be allowed; vegetables, on the other hand, must be given in somewhat larger quantities. The treatment of chronic constipation, one of the most constant symptoms, accompanying gastric atony, requires special mention. In the treatment the main reliance must be placed on the diet. Such forms of foods should be given that in the course of digestion produce substances to excite intestinal peristalsis; among these foods are Graham bread, certain vegetables, such as carrots, beans, tomatoes, peas, turnips, macaroni, stewed and raw fruits, buttermilk, honey, and cider. This form of diet will often overcome the constipation of atony without the aid of drugs. The following list has been found serviceable in the treatment of atony of the stomach:

		Calories.
7 A.M.	40 gm. orange juice	88
8 A.M.	200 gm. milk	135
	1 soft boiled egg	80
	60 gm. toast	154
	40 gm. butter	325
10 A.M.	100 gm. raw scraped beef	118
	60 gm. stale wheat bread	154
12 M.	100 gm. broiled steak	209
	or 100 gm. lamb chops (23)	
	or 100 gm. stewed chicken (106)	
	200 gm. asparagus	37
	or 100 gm. peas (318)	
	or 100 gm. spinach (165)	
	100 gm. mashed potatoes	127
	100 gm. apple-sauce	53
	50 gm. bread (stale)	130
3 P.M.	200 gm. milk	135
	60 gm. wheat bread	154
	40 gm. butter	325
7 P.M.	100 gm. boiled fish	80
	100 gm. milk	67
	60 gm. bread	154
	40 gm. butter	325
		<hr/>
		2850

In other cases of constipation, when the treatment just described proves ineffectual, injections of various kinds, especially of oil, may prove beneficial.

In atony of the stomach lavage is quite superfluous, and is not to be recommended. The use of the stomach douche is of greater benefit, especially in those cases depending upon the various gastric neuroses. Still more beneficial is the use of electricity, which may be applied either extraventricularly or intraventricularly by means of Einhorn's electrode. The best results are obtained by the intraventricular method. The tonicity of the muscular walls of the stomach is influenced by the faradic current. Painful conditions are alleviated by the galvanic current, the kathode being used intraventricularly, the anode placed upon the fundus of the stomach. To this may be added massage of the abdomen, the effect of which is to increase the peristalsis of the intestine and to strengthen the abdominal walls. In regard to the medicinal treatment, preparations of strychnine seem to serve the best purpose. Either strychnine sulphate or the extract of *nux vomica* may be given in pill form. To allay the feeling of pressure, which is a constant and annoying symptom, the extract of belladonna is prescribed. When there is a diminished secretion of hydrochloric acid, 15-drop doses of dilute hydrochloric acid, given according to the method of Ewald several times after meals, is indicated. In cases of increased acidity, sodium bicarbonate should be ordered after meals.

POLYSYMPTOMATIC NEUROSES. NERVOUS DYSPEPSIA (LEUBE) OR NEURASTHENIA GASTRICA (EWALD).

The polysymptomatic neuroses are characterized according to Boas, by the presence of an unusual multiplicity of symptoms, in contradistinction to the monosymptomatic forms, in which one symptom is prominent or alone

observed. The most important and typical example of a polysymptomatic neurosis is nervous dyspepsia, in which the symptoms are manifold and yet no organic changes can be detected. According to Leube's original conception, the term nervous dyspepsia was intended to cover that group of cases in which the secretory and motor functions of the stomach were normal while the patient was suffering from manifold subjective symptoms, following the ingestion of food. More recently Leube included in this class a group of cases manifesting changes in the gastric secretion and motility.

There are two forms of nervous dyspepsia: (1) Nervous dyspepsia in which no anatomical changes can be detected in the stomach. (2) Nervous dyspepsia in which the nerves supplying the stomach are involved in anatomical changes, together with consequent changes in the gastric secretion. Boas describes a third form originating "reflexly from other organs, such as from the kidneys, uterus, ovaries, male generative organs, and particularly in the intestines."

In addition constitutional diseases such as diabetes, tuberculosis, and syphilis may form a basis for the production of this condition. Nervous dyspepsia is also characterized by the fact that all the functions of the stomach, motor, secretory, and sensory may be disturbed at the same time. It is a well-known fact that nervous dyspepsia, accompanying organic diseases of the stomach, can be so combined with it that a most careful investigation may be necessary in order to differentiate between the primary and secondary affection.

Etiology.—As nervous dyspepsia is frequently associated with general neurasthenia, all conditions bearing an etiological relationship to neurasthenia are causative in the production of this disorder. A large number are due to worry, anxiety, reverses, overwork, sexual and alcoholic excesses, and also to the abuse of tea, coffee, and tobacco. Certain cases come from diseases of the blood, constitutional diseases or such other diseases as pulmonary tuberculosis and diabetes; other cases occur reflexly, the primary cause being eyestrain, genito-urinary disturbances, mental disturbances, or the puerperal state.

In this series of 1952 cases of functional gastric disorders there are 268 (16.5 per cent.) cases of nervous dyspepsia, 154 being males and 114 females. As to age, 33 were in the second decade, 55 in the third, 55 in the fourth, 68 in the fifth, 38 in the sixth, and 19 in the seventh. It occurs more frequently in males than in females.

Pathology.—In 41 patients suffering with nervous dyspepsia Jurgens¹ discovered a total degeneration of Meissner's and Auerbach's plexuses after death; so that in a certain number of cases there appears to be an anatomical basis for this condition. "Where the disturbance was more of a sensory character," there was, according to Jurgens, a degeneration of the muscularis mucosa of the stomach and of the intestines, also a pronounced formation of varices in the intestinal walls, the exact examination of which revealed a degeneration not only of the muscular fibers of the veins, but also of the sensory nerves and of the branches of Meissner's plexus in this vicinity."

Symptoms.—It is characterized by the multiplicity of its symptoms, varying greatly in intensity, mode of onset, and frequency. The most prominent sign is the gastric discomfort present after meals, although the

¹ *Verhand. des III Congresses f. Medicin*, S. 253.

distress is not dependent upon the quality and quantity of food ingested, but rather upon mental strain, worry, or excitement. It often happens that the most digestible food causes distress, while the most indigestible food is borne without discomfort. The gastric distress is characterized by pressure, fulness, eructations, distention, flatulency, peculiar sensations in the stomach, and heartburn. One food after another is discarded on account of the discomfort produced by eating until the condition of sitophobia is produced with symptoms of marked inanition. In addition manifestations of a general neurasthenia are often present, consisting of headaches, depression, vertigo, palpitation of the heart, lassitude, loss of strength, globus hystericus, and insomnia. The appetite is usually capricious at times, developing into a bulimia, and followed quite suddenly by complete anorexia. The general condition of the patient is not usually affected, although in some instances the emaciation may be so extreme as to suggest a serious organic disease. The periods of well-being, alternating with those of discomfort, are rather characteristic as also are the variations in the intensity of the symptoms from day to day. The abdomen is usually found sunken, occasionally distended with gas, accumulating often in the region of the cæcum and in the colon at the splenic flexure; the abdomen is often tender at the distended areas, but the pain is relieved by the expulsion of the gas. The bowels are, as a rule, constipated.

In the larger proportion of cases the gastric secretion is normal; occasionally there is a subacidity and less often a hyperacidity. A characteristic feature is the fact that when anacidity exists the ferments are not generally diminished in quantity; not infrequently marked variations in the state of the secretions are met with (heterochylia). In this series the gastric analysis showed normal acidity in 163, subacidity in 57, hyperacidity in 35, and heterochylia in 13.

As a rule, the motor function remains normal in this condition, although occasionally an atony of the stomach may occur. In the 268 cases a slight atony existed in 57; a gastropptosis is not infrequently found as a complication, it being present in 41 of this series.

Diagnosis.—This is made by the absence of all indications of organic disease, together with the general neurasthenic symptoms, associated with the digestive disturbances. It is further strengthened when the motor and secretory functions of the stomach are found normal. Greater difficulty, however, is manifested when these functions are not perfectly normal, but even then the symptoms present are so far out of proportion to the changes, that these slight variations will not account for the condition found. The discomfort is not dependent upon the quantity and quality of food, but is influenced by change of scene, diversions, etc. Indigestible food does not increase the distress.

It is always necessary to exclude all organic conditions before making a diagnosis of nervous dyspepsia. This is not always a simple matter. Boas advises the following plan in doubtful cases: A very easily digestible diet is given the patient, similar to that ordered during the third week of an ulcer treatment; while on this diet, the subjective symptoms are observed for a period of from three to four days, when other foods are gradually added, such as sauces, vegetables, desserts, and various fruits. The symptoms are now compared with those during the first period. In nervous conditions not only will they not increase in intensity on the fuller diet, but will often lessen,

while in cases of organic disease they will increase. Nervous dyspepsia must be differentiated from chronic gastritis, ulcer, carcinoma, and atony of the stomach. Nervous dyspepsia is distinguished from chronic gastritis in that the gastric disturbance is more or less directly dependent upon the quality and quantity of food ingested in the latter condition, while the gastric contents contains large amounts of mucus not observed in nervous dyspepsia. There may be an absence of free hydrochloric acid in both disorders, but an absence of enzymes does not usually occur in nervous dyspepsia. It is differentiated from ulcer of the stomach by the absence of a localized, painful area in the epigastrium which, although it may be present for a few days at a time is not constant; by the absence of hæmatemesis and occult blood in the stools and by the sudden and unaccountable change from severe gastric pain to the disappearance of all discomfort. Often the diagnosis can only be made after trying the effect of an ulcer cure.

In a certain number of cases of nervous dyspepsia the emaciation is so intense as to suggest carcinoma, especially if at the same time there is an absence of free hydrochloric acid. A correct diagnosis is arrived at by noting the age of the patient, the duration of the disorder, and determining the presence or absence of a motor insufficiency as well as of occult blood in the feces. The condition under discussion is further distinguished from gastric atony by the rapid changes in symptoms; and the non-dependence of discomfort on the quantity and quality of food ingested; atony and nervous dyspepsia are at times so combined that it is impossible to determine which is the primary disorder.

Prognosis.—This is not always favorable, although, as a rule, it is not a fatal disease. It may become rather obstinate and resist all attempts at treatment; the milder forms usually yield readily to proper treatment, while the severer ones continue over a long period of time before entire relief is afforded or may continue a downward path, with rapid loss of flesh and strength and finally end fatally. Even though the patient recovers, relapses are not uncommon.

Treatment.—Boas has made the statement that "in the larger proportion of cases of nervous dyspepsia drugs are not indicated. The patient should be taught that recovery cannot be brought about by the use of drugs, but only by a sensible mode of living and following the laws of hygiene." In all cases in which it is possible to determine the cause of the disorder, this should be relieved, *i. e.*, all intestinal disturbances should be corrected, anxieties and worries relieved, and sexual disorders treated. When no cause can be determined, the treatment should be directed toward the general neurasthenia. This is often best combated by change of scene and climate. The diet should not be too restricted, but strengthening food, without any attempt at a too rigorous diet, should be prescribed. In those cases in which milk is well tolerated it may be given in large quantities; when it is not well borne, buttermilk, kefir, or koumyss may be substituted. The patient's appetite is humored, and he may be allowed to eat any food he can digest. Alcoholic stimulants are prohibited, or given only in very small quantities. In severe cases a well-conducted rest cure will produce the best results. In those cases in which the patient has lost much flesh and strength, the most beneficial results are obtained by these means combined with forced feeding and isolation. Hydrotherapy, massage, and electricity together or alone, play an important role in the treatment.

Rosenheim has recommended the gastric douche in this condition, while others practice lavage. Of the medicinal remedies used the milder preparations of iron, arsenic, bromides, and the preparations of valerian must be noted. Mineral waters containing iron are sometimes valuable provided the waters are taken at the springs and proper hygienic conditions are maintained at the same time. The waters of Langenschwalbach, Franzensbad, Ritchfield and Rawly Springs are especially beneficial.

TABLE SUMMARIZING THE NUMBER AND PERCENTAGE OF EACH VARIETY
IN 1592 CASES OF GASTRIC NEUROSES.

MONOSYMPTOMATIC GASTRIC NEUROSES.

Irritative Group.

	Cases.	Per cent.	Males.	Females.
Superacidity or hyperchlorhydria	542	34.0	263	279
Supersecretion or gastrosuccorrhœa:				
Intermittent	21	1.5	15	6
Continuous	10	0.65	7	3
Digestive	2	0.10	2	0
Bulimia	24	1.5	5	19
Parorexia	8	0.5	2	6
Gastralgokenosis	7	1.5	7	0
Hyperæsthesia	31	1.5	4	27
Gastralgia	24	2.0	7	17
Rumination or merycism	24	2.0	20	4
Regurgitation	32	2.0	26	6
Eruetatio nervosa	54	4.0	12	42
Vomitus nervosus	49	3.0	15	34
Nausea nervosa	40	2.5	10	30
Cardiospasm	25	1.5	8	17
Pylorospasm	4	0.3	1	3
Pneumatosis	8	0.5	6	2
Peristaltic unrest	1	0.06	1	0
	906	57.61	411 (26 %)	495 (31 %)

Depressive Group.

	Cases.	Per cent.	Males.	Females.
Subacidity or hypochlorhydria	80	0.5	34	46
Anacidity or achylia gastrica	112	0.7	55	57
Akoria	5	0.3	3	2
Anorexia	48	3.0	11	37
Sitophobia	24	1.5	6	18
Incontinence of the pylorus	2	0.1	1	1
Atony	147	9.25	68	79
	418	26.15	178 (11 %)	240 (15 %)

POLYSYMPTOMATIC GASTRIC NEUROSES.

	Cases.	Per cent.	Males.	Females.
Nervous dyspepsia	268	16.5	154 (9.5 %)	114 (7 %)

CHAPTER V.

ORGANIC DISEASES OF THE STOMACH.

By CHARLES F. MARTIN, M.D.

GASTRIC AND DUODENAL ULCER.

Synonyms.—Simple ulcer of the stomach; round ulcer; peptic ulcer; chronic ulcer; perforating ulcer; *ulcus ventriculi* (Frank); Cruveilhier's disease (Cruveilhier was the first to give a good clinical and anatomical description of the condition).

The first name is doubtless the best, for these ulcers are not always round, neither are they always chronic, nor do they always perforate. Erosions of the gastric mucosa are probably often early conditions of gastric ulcer, both as regards etiology and morbid anatomy, and the subsequent auto-digestion affords the more typical appearance of the simple ulcer.

Definition.—A more or less progressive destruction, beginning in the mucosa and sometimes extending to and even through the deeper layers of the stomach wall, of the nature of a degeneration or a necrosis. These ulcers occur practically only where the gastric juice flows, and are found, therefore, only at the extreme lower end of the œsophagus, in the stomach itself, and in the portion of the duodenum above the opening of the common bile duct. Such ulcers may be acute or chronic, with or without tendency to cicatrization and healing. They leave an open loss of substance, usually round or oval in shape, with edges which are, as a rule, clean cut in the acute and subacute cases, more irregular in the chronic.

Einhorn and others do not regard this division into acute and chronic as justified, because, from a clinical examination, one cannot always tell, in the early stage, with which form one is dealing; in one sense all ulcers are probably chronic. There is some justification, however, for a clinical subdivision into the acute and chronic forms, the former having for its type the so-called acute perforating ulcer of young adults. Pathologically, the acute and chronic ulcers have the same etiology, and it is only the course which is different. Acute ulcers, such as occur in pyæmia, in portal thrombosis, and from trauma, are probably secondary to these processes, and thus of an entirely different nature.

Mayo has drawn attention to the fact that acute exacerbations of chronic ulcers are commonly mistaken for acute ulcers, and the subsidence of the acute period is often mistaken for the cure. "Chronic ulcer," he says, "is chronic from the start, and the early symptoms last from days to weeks."

Historical.—While it is true that records of patients with gastric ulcer are available from the sixteenth century (and records of probable cases even as early as the tenth century), and while these describe instances with hemorrhage, with perforation, and with fistula, yet it was not until Matthew

Baillie, in 1793, gave his accurate anatomical description, accompanied by clinical histories, that the disease possessed a definite historical entity.

Hunter advanced his anatomical theory of the causation about the same period, and already much attention was paid to both the clinical and the pathological features of the malady. Cruveilhier, however, was the first to give the subject a thorough investigation from all sides, and his *Anatomie Pathologique*, (1829 to 1835) gives most accurate and exhaustive descriptions of gastric ulcers. To him is attributable the rational distinction between cancer and ulcer of the stomach, and to him belongs the credit of giving to the subject its present status in medical literature.

Rokitansky in pathological observations and von Jaksch clinically did much to extend the knowledge of this subject. Brinton's analytical studies have likewise added much information to the literature, while the work of von Ziemssen, of v. Leube in medical treatment, and of Rydiger, Kroenlein, the Mayos, Robson, and Moynihan in surgery, have made important landmarks in the progress of the history of ulcer of the stomach.

Duodenal ulcer was described by Abercrombie in 1824, more definitely by Kraus in 1865, and more complete investigations were made by Chvostek in 1882 and Oppenheimer in 1891. The work of English and American surgeons on the diagnosis of duodenal ulcer and its treatment is worthy of special mention.

Statistical.—Statistics in regard to gastric ulcer are, on the whole, of very doubtful value. Those based upon *clinical* observation alone are unsatisfactory, because of the difficulties in diagnosis of obscure cases, and because many other conditions are often erroneously included. Statistics, again, bearing upon *pathological* examinations are often insufficient, either from the frequency with which simple ulcers are overlooked through lack of observation, or because the healing has been so perfect that often, as Bramwell states, no scar at all is left behind. The unsatisfactory grouping of cases of simple round ulcer with those due to causes of an entirely different nature, renders the statistics still more unreliable. *Combined clinical statistics* show 3036 cases diagnosed as ulcer among 339,575 patients, *i. e.*, 0.894 per cent. *Combined statistics of autopsies* of various series show 2608 ulcers out of 59,450 autopsies, *i. e.*, 4.4 per cent.

Geographical Distribution.—The regional differences are great, the most marked being, as Riitimeyer has shown, between North America on the one hand, and Central Europe and England on the other. Thus ulcer occurs as follows:

	Clinically. Per cent.	Pathologically. Per cent.
In North America	0.12 to 1.28 of all cases	0.85 to 2.35
In England	0.74 to 2.20 of all cases	1.20 to 4.60
In Germany	0.54 to 3.00 of all cases	1.30 to 11.00

It is thus much less common in North America, and statistics from South America and portions of Africa show the same infrequency. But, as Howard has shown, in North America itself there are again great regional differences. Clinically and pathologically it is commoner in the Northeast States than in the South (excepting San Francisco, which has the highest percentage, 2.35), but even this last is less than half the incidence in European statistics, in which in Brinton and Lebert's series it averages about 5 per cent. In Europe, Russia shows few cases. In the St. Petersburg statistics (pub-

lished in 1886 to 1887) there were no cases of ulcer among 3750 admissions, and the autopsies likewise showed it to be of great rarity, viz., 0.5 per cent. In Denmark, on the other hand, it is much more frequent (13 to 20 per cent.). In Germany, according to von Sohlern, there are few cases in the Rhone Valley and the Bavarian Alps, while at Kiel and at Thüringen it is found in 8 to 10 per cent. of the autopsies. It is doubtful if these differences are determined, as von Sohlern thought, by the extra vegetable diet in these places, the ulcer being supposedly prevented by the potassium therein. The theory does not hold good on close scrutiny. One general rule obtains in Germany, viz., that, as one goes from North to South, ulcer is less frequent. The average of pathological statistics (Rütimeyer) shows: Russia, 0.8 per cent.; Switzerland, 2.6 per cent.; Austria, 4 per cent.; Germany, 5 per cent.; England, 5 per cent.; Denmark, 16.7 per cent.; and North America, 1.3 per cent.

Differences exist, too, for individual cities, as the following reports indicate: *Clinically*, Cantlie's statistics from the Royal Victoria Hospital, Montreal, show that gastric ulcers form 0.004 per cent. of all cases admitted and 0.97 per cent. of all medical admissions. C. P. Howard, in his collected statistics for America, shows that out of 161,589 medical admissions in fifteen hospitals, gastric ulcer was present in 0.57 per cent. In Berlin, the statistics for ten years, from 1888 to 1898, showed 1.33 per cent.; that is, 0.76 per cent. higher than Howard's. In Edinburgh they formed 2.02 per cent. of all admissions, and in London only 0.78 per cent.

Pathological statistics show likewise a great variation. In Thüringen, Miller and Starcke observed it in 10 per cent. of autopsies, and Grünfeld found it in 20 per cent. of the Copenhagen autopsies. Keating, in 1881, gave 0.09 per cent. as the cause of death in New York City in 444,564 deaths, while Welch's statistics, on the other hand, are 0.75 per cent. The average in Europe is much higher, 5 per cent. and more. In Berthold's Berlin series (262 cases) ulcer was present in 2.7 per cent. of all autopsies. The recent Basel statistics of Rütimeyer show 2.06 per cent. in 3000 autopsies. In 47,912 London autopsies Fenwick found 2079 ulcers, which is equal to 4.2 per cent., while in Europe there were 8.54 per cent.

As a cause of death ulcer occurred in 410 out of 444,564 deaths.

Etiology.—Simple ulcer of the stomach has as its main etiological factors a destruction of gastric epithelium, disturbance of the circulation in the stomach, and acidity of the gastric juice, the combination permitting auto-digestion.

Age.—Gastric ulcer is commonest between twenty and thirty years of age, though the age incidence differs according to the sex. The majority of cases of ulcer occurs in males from forty to forty-five years of age, and in females from fifteen to thirty years. Combined statistics show the average for males to be 36.7 years, and for females, 27.1 years of age. At the Royal Victoria Hospital, Montreal, the average age for gastric ulcer was twenty-seven and a half years. In infancy it is rare, no case occurring under ten years in 262 autopsies on cases of ulcer at the Berlin Pathological Institute. Cutler studied 29 cases, of which 6 were found immediately after birth, 8 under seven years of age, and 9 between the ages of eight and thirteen years. Rehn, on the other hand, collected 9 cases of simple ulcer in children, and Kundrat speaks of having frequently found minute pinhead-sized ulcers in children, which, however, were probably the result of hemorrhage,

and differed thus from ordinary round ulcer. Martha Wollstein, Holt, and others have added cases, though many experienced pediatricists have never observed a case clinically. The symptoms seem less distinctive than in adults, and depend chiefly on evidences of blood in the vomitus or feces, for which many other causes than ulcer may be found, *e. g.*, hemorrhages from the nose, throat, lungs, or from the mother's breast, or again from general constitutional diseases, such as scurvy, purpura, the malignant exanthemata, cirrhosis of the liver, etc. Perforation seems to be relatively more common in children than in adults. Henoch, too, found ulcers not rarely in the newborn, and, according to Osler, they are sometimes found in the foetus.

An example of the presence of ulcer at the other extreme of life is reported by Eleanor C. Jones, in a man aged eighty-four years, with a past history of gastric ulcer thirty years before, and in whom, at death, a fresh ulcer was found next to the scar of the old healed ulcer. Riegel's statistical table (compiled from clinical cases) is interesting:

Riegel's table.				The writer's combined clinical statistics.		
Age.	Men.	Women.	Total.			
0 to 10 years	1	0.14	per cent.
10 to 20 "	8	35	43	85	12.00	"
20 to 30 "	29	62	91	259	37.00	"
30 to 40 "	35	22	57	186	26.50	"
40 to 50 "	36	11	47	110	14.00	"
Over 50 "	18	4	22	63	9.00	"
Total	126	134	260	704		

The pathological statistical table of open ulcer gathered from the writer's combined statistics is as follows:

Age.	No. of cases.
0 to 10 years	1
10 to 20 "	49
20 to 30 "	163
30 to 40 "	134
40 to 50 "	142
50 to 60 "	129
60 to 70 "	102
70 to 80 "	41
Over 80 "	7
Total	768

In Lebert's statistics the mortality was greatest between forty and sixty years of age. According to Fenwick, 70 per cent. of the ulcers under thirty years of age are acute, and only 7 per cent. are chronic.

Sex.—Statistics on this point vary greatly, and one should not attach too much importance to individual reports. Clinically, most statistics show preponderance of women over men, in the proportion of 2 or 3 to 1, but pathologically the numbers are about equal in both sexes. The writer's *combined statistics* of all series give, clinically, 1 man to 2 women, and pathologically, 1804 men to 2912 women.

Whether gastric ulcer is more frequent in men than in women is apparently a matter of chance, so far as our present knowledge goes. Thus while Habershon found, in 201 cases, 127 in women and 74 in men, Lebert,

in Breslau, found ulcer in the proportion of 3 or 4 men to 1 woman, while Riegel, clinically, had 126 ulcers in men and 134 in women. So, too, in Basel the autopsies show equal numbers in both sexes. Friedler's pathological statistics of 2200 cases give 20 per cent. in women and only 1.5 per cent. of ulcers in men; Berthold's series, 128 men to 134 women; in Grünfeld's high percentage there were 241 women to 209 men; Brinton found ulcers in 2 men to 1 woman; and Danziger, vice versa.

Occupation.—This has a doubtful relation to ulcer, and no walk of life appears to be exempt. According to some authorities, cooks are predisposed, and presumably because of their habit of swallowing foods which irritate, from one cause or another. In Payne's statistics only 3 out of 50 females were cooks. Workers in metals (Bouveret), glass and porcelain factories (Bernitz), who are in the habit of swallowing dust particles, are thought by some to be more liable, so also tailors and shoemakers, because of their position and occupation leading to abdominal pressure.

Trauma.—As a cause of gastric ulcer, this may be external or internal. Internal trauma, arising from hot foods, corrosives, foreign bodies, and mechanical and chemical irritants, may of course produce gastric ulcer, but the character of the lesion is often entirely different from that of simple round ulcer. External trauma, arising from injuries, has been frequently recorded as a cause. The injury may be a blow or tight-lacing, ill-fitting corsets, or from compression, as in the case of tailors and cobblers. Ackermann, studying the material from P. Cohnheim's Berlin Policlinic, concludes that epigastric compression is by no means an uncommon factor in the causation of ulcer, and for such conditions as these the term "occupation compression" is used. Burns of the skin are indirectly related to gastric ulcers.

Ritter produced experimental submucous hemorrhage in dogs after inflicting blows on the stomach, in one case causing even a separation of the mucous membrane, and it was his opinion that injuries predisposed to ulcers through the subsequent action of the gastric juice. He further describes an incident in which injury in the epigastric region was followed in eight days by hæmatemesis and other signs of gastric ulcer. Fertig describes an apparently undoubted case in which, as a result of a kick in the abdomen, four gastric ulcers were found at autopsy. Nevertheless, as J. MacFarland points out, such ulcers usually cicatrize quickly, taking quite another course from ordinary simple ulcers. One may conclude, however, that such injuries sometimes provide the impetus for the later formation of true ulcer, and probably the mucous membrane is separated from the wall and necrosis occurs, thus starting the ulcerating process. The same probably holds true of the theory of Rokitansky and Rindfleisch and Key, that ulcers arise from hemorrhage and subsequent erosion. The two processes seem different, but erosions, once formed, are in themselves perhaps not infrequently followed by simple ulcer, the stimulus being given by bacteria or chemical action.

This seems to be the view taken by Richardière, whose traumatic ulcer cases are classed in two groups, the one, acute cases, healing rapidly, the other, cases running a course similar to ordinary simple chronic ulcer. The difference in the result may depend on the condition of the gastric juice, especially as regards its acidity.

Violent emesis as a mechanical factor in the causation of gastric ulcer is

mentioned by some, but is often, doubtless, associated with underlying causes, such, for example, as uræmia.

Disease.—*Chlorosis* and *anæmia* are thought by many to be predisposing factors, and, vice versa, chronic ulcer certainly often leads to *anæmia*. Samuel and W. S. Fenwick hold that a definite relation exists between ulcer and *anæmia*, and 72 per cent. of their cases showed symptoms of *anæmia* before serious ulcer symptoms developed. Riegel, too, regards *chlorosis* as primarily inducing hyperacidity, which in turn helps to cause gastric ulcer. Fitcher examined 44 cases, and found the average to show a chlorotic *anæmia*. The red cells numbered 4,071,000, the white cells 7500, and the hæmoglobin was 58 per cent. After hemorrhage there was often leukocytosis, while the digestive leukocytosis was slight. Perhaps, as Cabot and Stengel suggest, the *chlorosis* is often due to the hemorrhages, though in Fenwick's case the *chlorosis* occurred without these. Mayo regards *anæmia* as an important cause, and believes that by treating the *anæmia* one can often cure the ulcer. There can be little doubt that ulcer occurs more commonly among those who are *anæmic* than in persons in perfect health.

Arteriosclerosis and *endocarditis* seem at times to be etiological factors. In Howard's cases, 48.8 per cent. had sclerosed vessels, and in 22 per cent. the condition was marked. The Basel clinic showed 14.5 per cent. to have some disease of the circulatory system.

Previous gastric trouble as an etiological factor is hard to estimate. Mathieu, Galliard, and others regard gastritis as important, especially if combined with hyperacidity, all the more so if localized inflammatory plaques exist associated with it. It is difficult to draw the line between the symptoms of slight gastric disturbances and the early signs of ulcer, so that a supposed preliminary gastritis may merely have been the indefinite symptoms induced by the already existing ulcer apart from any inflammation of the stomach. The researches of C. H. Miller would indicate that inflammatory swelling and rupture of lymphoid follicles may induce erosions which possibly develop later into true ulcers, and in this way, it is true, a definite relation might be said to exist between gastritis and ulcer.

Dietetic Errors.—Dietetic errors are scarcely a factor per se in the causation of true gastric ulcer. Neither alcohol (despite the views of G. de la Tourette and others) nor coarse vegetables and indigestible foods can be regarded as frequent antecedents; nor, again, are poisons in themselves a cause.

Infections.—Letulle found gastric ulcer in a man suffering from dysentery, and isolated bacteria from the stools, which when injected in pure cultures into guinea-pigs produced specific erosions and ulcerations. He was of the opinion that some ulcers, at least, might be due to bacterial invasion. Chantemesse and Widal produced typical perforating ulcers by introducing the bacillus of dysentery directly into the stomach of guinea-pigs. Erosions and ulcers are frequently found in general infections, *e. g.*, pyæmia, but in most cases of true ulcer bacterial invasion is scarcely the cause, though Martin believes the pyloric site of ulcer favors the bacterial theory, because these parts are less subject to the antiseptic action of the gastric juice.

Syphilis, by producing vascular changes in general, may predispose to gastric ulcer. There is no relation between the specific syphilitic gastric ulcer and simple round ulcer. There was a previous luetic history in 10 per cent. of Engel's cases and 20 per cent. of Lang's.

Tuberculosis as an etiological factor has an extremely doubtful relation to gastric ulcer, although, of course, tuberculous ulcers of the stomach, as evidence of the specific infection, are no longer rarities. They are usually multiple. Perhaps 20 per cent. of patients with ulcer die of tuberculosis. In 6 out of 22 fatal open ulcer cases in Montreal, pulmonary tuberculosis existed, but in over half of these it had no relation to the cause of death.

The same may be said of trichiniasis and infections generally, which are a frequent cause of gastric ulcers of another type. Round ulcer is a pure necrosis, and is not an infection so far as our present knowledge is able to demonstrate.

Pathogenesis.—The following facts may be accepted as proven

1. Gastric ulcer occurs wherever gastric juice flows.
2. The process in its earliest stages is essentially a necrosis.
3. Healing is prevented in some way by the action of the gastric juice.

The relation of trauma and infection has already been discussed.

The Relation of Hyperacidity to Gastric Ulcer.—One cannot accept the statement made in recent years, that hydrochloric acid is regularly increased in gastric ulcer, and that statistics vary so greatly in this particular must be due either in part to chance or to varying ideas as to what constitutes hyperchlorhydria, what means are employed in testing, and by what color reactions, etc., one should be guided. Perhaps, too, regional differences are of some importance. Recently, Ewald, out of 132 cases found hyperacidity in 34.1 per cent., subacidity in 9 per cent., and normal acidity in 56.8 per cent. Lenhartz, out of 13 cases, found hyperacidity in only 3, and subacidity or anacidity in 5. Wirsung found 42 per cent. of his cases with hyperacidity, and Wagner an equal number. Einhorn found in America that most of his ulcer cases showed hyperacidity, as also did Robin in France and Fenwick in England (75 per cent.). Riegel's original statement, therefore, that hyperacidity is the rule, can scarcely be accepted in view of subsequent analysis. Rüttimeyer, in 162 cases (36 men, 126 women), found 42.5 per cent. with hyperacidity and 57.4 per cent. with the acidity normal or diminished. He found hyperacidity relatively much more frequent in men.

The following statements may be regarded as justifiable in the present state of our knowledge:

1. Gastric ulcers occur only in those regions where hydrochloric acid flows; hyperacidity is common in gastric ulcer, though in many cases there is no increase of hydrochloric acid; in fact, there may be anacidity. The amount of acid in cases of ulcer varies, too, at different stages and periods of the malady. It is probably greater at the time immediately preceding or following a hemorrhage. Many exceptions to this occur.

2. Hyperacidity is common in the absence of gastric ulcer.

3. In itself it is insufficient as a cause to produce gastric ulcer.

4. It occurs commonly in chlorosis, and thus may be an additional indirect factor in the etiology.

5. Hyperacidity may induce gastric ulcer, when through any cause a portion of the mucous membrane is injured, and by virtue of the acid the ends of any exposed vessels are contracted, thereby inducing local anæmia and necrosis.

6. Hyperacidity prevents healing of gastric ulcer, as has been demonstrated by Matthes in his experiments on dogs, in which traumatic ulcers of

the stomach were prevented from healing by irrigation with 0.56 per cent. solution of hydrochloric acid. In these cases hyperacidity does not form ulcers, but causes them to become chronic. Richardlière divided traumatic gastric ulcers into two classes: (a) Those which heal quickly because the gastric juice is normal. (b) Those which heal slowly and form typical simple ulcers because hyperacidity exists.

7. Secondary ulcers which form after gastro-enterostomy are probably associated with excessive hydrochloric acid secretion.

8. Hypersecretion sometimes occurs in gastric ulcer, and in such cases the hyperacidity is especially great and constant.

The Circulatory System in Relation to Ulcer.—Various conditions are regarded as possible factors aiding in ulcer formation, as was mentioned by Morris in 1800. The main features are stasis or local anæmia of the gastric wall, allowing the gastric juice to digest the part thus rendered less resistant.

1. Virchow regarded *embolism* as one factor, though not the exclusive one. It was found that in heart lesions, etc., emboli existed and seemed to cause ulcers, as they do infarcts in other organs, and again, where ulcers existed without other lesions, emboli were sometimes found to correspond. Orth regarded the oval, oblique shape of many ulcers as proof, too, of this theory, the shape and site corresponding often to arterial distribution. Experimental proofs have also been brought forward, *e.g.*, Panum injected wax emulsions into the general arterial circulation (femoral) of dogs. Cohnheim did the same with chromate of lead injected locally into the coronary arteries of the stomachs of dogs. Each experimenter in this way produced gastric ulcers.

Against Virchow's embolic theory it may be argued: (a) Cohnheim showed that gastric arteries are not terminal, that a large capillary network terminates the arterial supply, that necrosis could only occur, therefore, in a very limited area of mucous membrane. Embolism in such a way is, therefore, exceptional. (b) Emboli are not always found. (c) Ulcers occur most frequently when no cause of embolism exists, and in young people. Ligation of the arteries of the mucosa produce ulcers, but not so with ligation of the trunks outside of the mucous membrane.

2. Virchow added *thrombosis* as an additional predisposing cause. The same line of argument holds here, for while gastric arteries do often show endarteritis even with obliteration, yet this is rather a result than a cause of ulcer, and in any case, were this a factor in the causation, the extent of the lesion would be much larger, as the arteries have such extensive collaterals.

3. *Hemorrhagic Erosions and Submucous Hemorrhages.*—Erosions are common in the stomach, most of all near the fundus, and are small, multiple, and usually hemorrhagic. At other times they are abundant at the pylorus. They are either superficial or involve the submucosa, show a bloodstained, grayish-red floor, well-defined edges, and are more or less circular or oval in shape. Around them the immediate vicinity of the mucosa usually shows hemorrhagic infiltration. They seem to originate from capillary hemorrhages or tiny infarcts of the submucosa.

These erosions are very common, and are frequently associated with chronic heart, liver, or kidney disease, and with the acute infections; they occur likewise in various cachexias and in the newborn. Postoperative hæmatemesis is usually due to these erosions. The condition is certainly not confined to operations upon the appendix, as was believed in France

(vomito-négro-appendiculaire). Many regarded these erosions as practically similar to the ordinary acute gastric ulcer.

Authors differ as to their relation to ulcer. While, for instance, Langerhans thinks, on account of their site, number, and confluence, that they are rather associated with inflammatory lesions and spastic contractions than with gastric ulcer, Gerhardt and Namverde regard them as frequent antecedents. The former, indeed, regards many of them as true ulcers from ruptured swollen follicles, and Gaillard and others have called attention to the frequent small round-cell follicular inflammation found rupturing toward the mucosa. C. H. Miller expressed a similar opinion, as has already been mentioned.

It would seem to be an easy matter to produce mere erosions, for various experimenters have induced the lesions in a great variety of ways. Schiff and Ebstein found them after injuries to the brain, Ewald after section of the cord, Talma by increasing the tension on the stomach wall, and Müller by ligating the portal vein, each method yielding the experimental erosions. Ritter, again, found the more direct injuries to the stomach wall capable of producing the lesion, and especially if, after inflicting blows on the abdomens of the animals, he introduced hydrochloric acid into the stomach.

Matthes endeavored to show that some relation existed between defects in the mucosa and true ulcer by a series of experiments, the fundamental idea of which was that ulcer followed erosions unprotected by surrounding folds of mucous membrane. He removed bits of mucous membrane, and found that there was a tendency of the muscle in these regions to contract and thereby bring overhanging portions of mucosa together to cover over the defect and thus protect the bared portion against the action of the gastric juice. Where, by artificial means, this contraction was prevented, the hydrochloric acid would tend to prevent healing and would gradually induce a true ulcer, all the more so if excess of acid were introduced. Where contraction was prevented over unbared portions of mucosa, no ulcer appeared, even if great excess of hydrochloric acid was introduced.

Bloch, in his experiments along these lines, maintained that the pylorus and lesser curvature, being less subject to contractions and having less heavy folds of mucous membrane as a protection, naturally were the commonest sites of ulcer. He thought that the erosions due to nerve lesions were largely explained from the fact that, as a result of the injury, muscular contraction of the stomach wall was lessened, and thereby the folds of mucosa could not protect the bared surface.

One of the great objections to these theories lies in the fact that ulcer is by no means common in gastrectasis, where contraction of the muscle fails. A. Schmidt, and also Griffini and Vassale, believed muscular contractions had much to do with their development and healing according to the amount of protection afforded by the mucous membrane. Still injuries of this kind to the mucous membrane usually heal quickly under ordinary conditions, and chronic round ulcer is not the type usually found as a result. At most, such injuries may develop later into true ulcers.

4. *Arterial spasm*, either by itself or from muscular contraction of the walls acting on the traversing arteries, was thought by Klebs to be a cause of ulcer; but this is probably not the case and sufficient proof is still lacking.

5. *Diminished Alkalinity of the Blood*.—Pavy some years ago advanced the theory that normally the mucosa of the stomach is prevented from necrosis

or digestion by the circulating blood; that the virtue lay in its alkalinity, which prevented all cells in contact with it from being digested by the gastric juice with its acid, *i. e.*, neutralization occurred. "Diminished alkalinity of the blood favored autodigestion, because the action of the acid was not neutralized." Against this it may be argued that the lesion is local, while the effects of diminished alkalinity are necessarily diffuse. It is not proven that the cells are alkaline; even although surrounded by alkaline blood, or that with embolism, etc., in the stomach, it is the absence of alkalinity which favored digestion of the mucous membrane. Probably the condition is merely an anæmic necrosis.

Again, the amount of alkalinity is small as compared to the degree of acidity. Samelson's experiments show the relative unimportance of the chemical composition of the blood, because, by introducing large quantities of acids into the stomachs of animals, and even into the gastric vessels, he yet never obtained autodigestion, as long as the blood could flow unimpaired through the vessels of the stomach wall. Jaksch again found alkalinity of the blood in chlorotics, so that its effect, if present, would be at most indirect.

Neuropathic Theory.—The effect of an injured nervous system in producing gastric ulcer has received consideration from many authors and much has been done in an experimental way to show a relationship. It may be said, however, that beyond lowering the resisting power of the tissues of the stomach, and also inducing hyperacidity, there is little connection between injury of the nervous system and the formation of gastric ulcer. Years ago Brown-Séquard found ulcers developing after excision of the corpora quadrigemina, and Schiff, Ebstein, Ewald, and others produced similar results by various injuries to the brain.

Talma faradized the left vagus to obtain spasmodic contractions of the stomach, then added hydrochloric acid solution over the mucosa and obtained gastric ulcers; and Yzerin got similar results in 10 out of 20 rabbits in which he cut the vagi below the diaphragm. Some even perforated, and he believed the results were due to spasm of the pylorus followed by interstitial hemorrhages and subsequent corrosion by the gastric juice. Dalla Vedova again found that injuries of the celiac axis and splanchnics caused hemorrhagic erosions of the stomach and necrosis in nearly half of the rabbits experimented upon, although he obtained negative results on injuring the vagus. Donati's experiments failed to confirm Vedova's results.

MacFarland's Conclusions are Interesting.—He regards as essential features the corrosive action of the gastric juice, which is merely an accident acting upon a more important feature, namely, the diminished resistance locally of the tissues, due to some change nutritional and vascular, whether depending on injuries of the small vessels from overdistention, pressure, embolism, thrombosis, infection, intoxication, or defective innervation; which or how many of these may be present is still an open question. The nature of the lessened resistance is unknown, just as is the nature of immunity of living tissue to its own products. Weinland's recent view, based on studies of immunity, regards ulcer as a destruction of epithelium due to the result of failure in these cells to produce an antibody which antagonizes the digestion of vital tissues by the ferments, *i. e.*, an antipepsin; this failure may be the consequences of hyperacidity of the media, or else the destruction of an antipepsin in hyperacid media. But here, again, if this be the case, why should

the lesion be only local, and secondly, it is not the pepsin, but the hydrochloric acid, which does the harm. MacCallum has suggested that all tissues may have an antiferment, which is specific for the individual or the species, against a digestive ferment of the same species.

Morbid Anatomy.—Situation.—These ulcers exist wherever the gastric juice flows. The posterior surface of the stomach is the commonest site, and ulcers are found there in 42 per cent. of all cases, especially near the pylorus and along the lesser curvature. The situations are variously described. Welch's statistics of 793 cases and those from collected tables combined are compared below:

	Welch's statistics.	Combined statistics.
On the lesser curvature	288 or 36 per cent.	726 or 35.0 per cent.
Posterior wall	235 or 30 "	585 or 28.0 "
Pylorus	95 or 12 "	291 or 14.0 "
Anterior wall	69 or 9 "	182 or 9.0 "
Cardia	50 or 6 "	134 or 6.5 "
Fundus	29 or 4 "	62 or 3.0 "
Greater curvature	27 or 3 "	75 or 3.6 "
Anterior and posterior walls together		14 or 0.67 "
Total	793	2069

Brinton's 206 cases show some variation:

The posterior wall	42.0 per cent.
The lesser curvature	26.8 "
The pylorus	15.6 "
The anterior and posterior wall	4.0 "
The greater curvature	2.4 "
The cardia	2.0 "

Fenwick's statistics give:

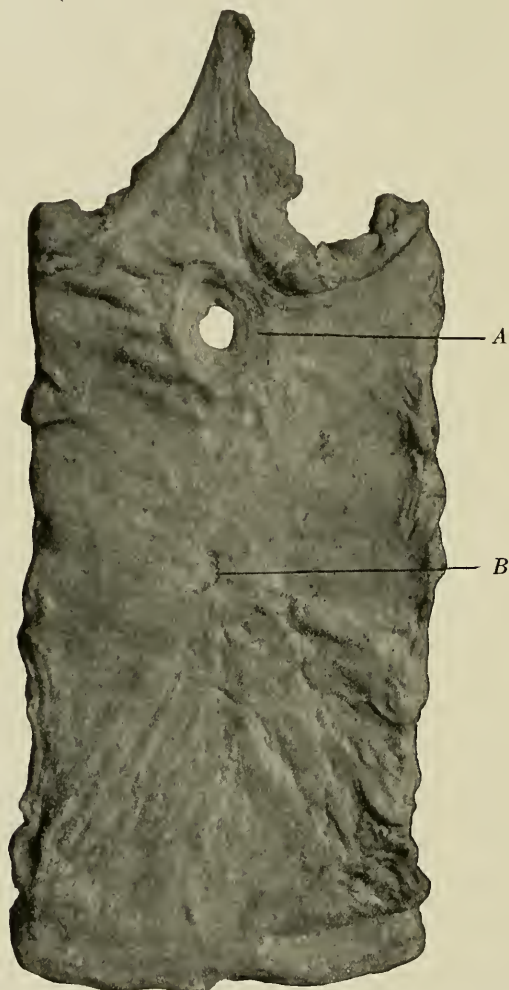
	In 70 chronic ulcers.	In 39 acute cases.
Pyloric region	53	13
Middle zone.	7	14
Near the cardia	10	12

Number.—These ulcers are usually single, but often multiple. Indeed, that they are commonly multiple is becoming more and more recognized. In one of Berthold's cases there were 34. Sometimes a perforated one is found at operation, and after healing another far distant may perforate. In Brinton's 463 cases, 381 had one ulcer, 57 showed two ulcers, 16 had three, 3 had four, 2 had five ulcers, and 4 had more than five. When multiple they may all seem to be of the same duration, or some may be in a state of healing, while others seem to be quite recent. Welch and others regard 20 per cent. of cases as multiple; Carless thinks acute ulcer is multiple in 50 per cent., while, according to Fenwick, chronic ulcer is multiple in only 13 per cent. In 13 out of 21 cases operated on and resulting fatally, Carless found more than one ulcer. Sometimes one ulcer is in the stomach and another in the duodenum.

Shape.—The more acute ulcers are punched out in appearance, round or oval and sharp in outline, while the less acute ones are shelved or terraced, the widest diameter and the greatest loss of substance being at the mucosa. The ulcers become gradually narrower as one approaches the serosa (funnel

like). This was seen to correspond to the distribution of the bloodvessels, and Orth has already drawn attention to this fact. The still older lesions are often irregular in outline, spread out, and with the mucosa rolled inward and thickened at the edges. If extensive, they are serpiginous, and this is

FIG. 1



Two gastric ulcers: *A*, showing a shelving base and central perforation; *B*, more superficial.

especially the case where several have fused. Thickening and raising of the edges occur as the ulcer gets older. Often they are saucer-like, with bevelled edges, and rounded out or concave.

Cicatrization may often be seen progressing in various portions of the walls. The muscle beneath is shrunken, the floor is smooth and grayish

brown or red, rarely ragged. It is generally covered with some necrotic tissue, and in old ulcers with indolent granulation tissue. Sometimes there is a hemorrhagic exudate.

Adhesions may occur to any surrounding or neighboring tissue or organ.

Size.—The usual size varies from 2 cm. in diameter to 3 to 4 cm.; in many instances, however, they may be as small as 1 cm. or less, or, again, as large as 10 cm. in diameter, sometimes even forming a ring, more or less completely surrounding the organ. Peabody records one measuring 19 by 10 cm.

Microscopic Examination.—The lesion is essentially a necrosis. There is very little inflammation or sign of healing in acute round ulcer, and even in many older ones; hence the slow repair, resembling somewhat the *ulcus perforans pedis*. There are, moreover, some signs of digestion; bacteria and moulds are also often present.

Healing and Cicatrization are Common.—Undoubtedly many ulcers occur and heal without giving symptoms of any kind. Many are superficial erosions rather than ulcers and healing occurs with granulations from the submucosa and scar formation. At all events, the frequent presence of scars and little fibroid areas suggests the possibility of ulcer as the cause of these. The healing process commences from the sides and floor of the ulcer. There is proliferation of the adjoining fibrous and glandular tissues, the edges become indurated, and the margins are more indefinite and irregular in outline, with signs of puckering and contraction. The gland structures are repaired to a limited extent only; the new tubules are small and atypical, not like the original epithelial structures, but with cylindrical cells often arranged around the lumen, with no opening toward the stomach cavity and no secretion from the cells. The granulations are usually indolent, the healing slow and irregular, rather than showing well-defined scars. Sometimes in old ulcers the healing process leaves chronic inflammatory areas which tend to erosion and recurrent bleeding. This may be severe and even fatal if large vessels are involved. The area of stomach around old ulcers is often infiltrated to a great extent with small cells and fibroblasts. Where muscle tissue is lost, it is replaced by fibrous tissue.

Scars vary in size, and according to their size and situation and to the amount of contraction which follows the healing process, they may or may not cause perceptible deformities in the organ and corresponding functional disturbance. The deformities thus caused comprise hour-glass contraction, pyloric stenosis and gastrectasis, and other irregularities with or without obstruction.

Perforation occurs when the ulcer penetrates down to the serosa before adhesions have formed, and for this reason it naturally occurs most commonly on the anterior wall.

Surgical experience does not always show that hyperacidity prevents healing, and after many operations on stomachs with increased secretion of hydrochloric acid healing results without trouble.

Symptoms.—Latency.—There may be no characteristic symptoms, and the first discovery of a gastric ulcer may be made unexpectedly at autopsy. Stoll found it latent in 27 per cent. of the cases in which it occurred in 3476 autopsies; Savariaud in 20 per cent.; Lebert found that 15 per cent. of 110 cases were latent until severe symptoms arose. Then, again, the first evidence may be a sudden or perhaps fatal hemorrhage, or the peritonitis following a

perforation may give the first indication to the patient or physician that ulcer has been present, even after months of what seemed an ordinary chronic dyspepsia. The symptoms may simulate spinal disease, hysteria, hyperacidity, and only upon the appearance of more serious signs does the diagnosis become clear. It may be latent, even if perforation has taken place, as in one instance in which an ulcer giving suspicious signs of perforation remained quiescent. The patient for some days after the event seemed to improve, and there was little or no tenderness in the abdomen. Several weeks later an abscess developed in the pelvis, which was opened per vaginam and drained, and the autopsy revealed the origin to be in a fistulous opening from a perforated gastric ulcer. Ashhurst records a similar case of interest.

Sometimes nothing but backache occurs, as in the case of Beneke, where for years this was the only symptom; a sudden peritonitis, however, from perforation, caused death, and the autopsy showed old adhesions of the liver and a perforation, between the adhesions into the abdominal cavity. Sometimes spinal disease is suspected, and a plaster jacket has even been put on.

General Symptoms.—*Dyspepsia.*—While the cardinal signs are pain after eating, epigastric tenderness, vomiting, and hæmatemesis, yet often, perhaps even as a rule, the symptoms are less definite and dyspepsia is the only evidence to be found, with perhaps a little vomiting or regurgitation. There may be a sense of fulness, weight, distention, and gaseous eructations. With these there may be pyrosis, occurring during the digestive period, or there may be mere gnawing or burning or "*heissshunger*" (from hyperacidity). Orthoform has been used as a test, dulling sensitive exposed areas of ulcer and having no effect on mere hyperæsthetic mucous membranes. Its use, however, has justly been called into question, as its action is unreliable.

The *general condition* of the patient, as a rule, remains good, although *anæmia*, *emaciation*, and *weakness*, all due to local causes in the stomach, are common and sometimes marked. *Nervousness*, *melancholia*, *headache*, and *dizziness* are frequent; so, too, are *amenorrhæa* and *dysmenorrhæa*. *Fever* is usually absent, unless complications arise (peritonitis, hemorrhage, etc.). Often there may be a rise of 1° without complications, perhaps associated with *anæmia*. The appetite varies; it may be excessive, but the patient fears to eat because of pain. *Thirst* is common. *Constipation* is the rule because of various factors, *e. g.*, sex, food, *anæmia*, lack of exercise, and vomiting. The stools may show blood.

The *urine* is lessened in quantity when vomiting occurs or when less nourishment is taken. It is usually less acid than normal, especially when gastric hyperacidity is present. Indeed, it is often alkaline, more particularly if gastrectasis be present, and then, if there be fermentation in the stomach, the urine will show excess of ethereal sulphates and indican. Acetone and diacetic acid are sometimes present, and one may even have serious acidosis. Other alterations are unimportant. Sometimes there is albumin, and, as has been shown by von Noorden, not uncommonly after severe pain. Dreschfeld found albumoses occasionally present.

Special Symptoms.—*Pain.*—The pain in gastric ulcer is one of the most prominent features of the symptom complex, and is variously described as boring, burning, gnawing, cutting, and tearing, rarely lancinating or cramp-like; sometimes merely a dull ache, paroxysmal in character, and at times

excruciating; not infrequently it radiates to the back. Sometimes it is a mere burning sensation, especially with an empty stomach, and is often relieved by food. Pain occurs in over 90 per cent. of all cases.

Time of Appearance.—It occurs during the digestive period either at once after taking food, or at the height of digestion; the hyperacidity increases it. When the ulcer is at the pylorus the pain may not appear for from three to four hours after meals, this being about the only condition in which the time of the pain gives a clue to the location of the ulcer. Riegel lays stress on the *site* of the pain as an important feature because he found left-sided gastric pain, which is constant or intermittently progressive, to be a sure sign of ulcer. Pain is found under the costal edge, at first only slight and paroxysmal, but later on it becomes more constant and is accompanied by vomiting. This gives a clue to the early diagnosis before cicatrices form or abnormal shapes occur. The features of this left-sided gastric pain are these: there is an absence of any other cause for it; it is not a reflex pain, because such pains are situated in the median line or are right-sided; and again, it is constant, worse after meals, always in the same spot, and gradually leads to emesis. The onset of the pain three or four hours after meals is also common to duodenal ulcer.

Effect of Food upon Pain.—It is distinctly aggravated by a large amount of food or by food that is solid, but there are many exceptions, and food sometimes gives relief, especially albuminous food, if there be hyperacidity. Sometimes, too, there is no relation whatever between the food ingested and the onset of the pain. Rüttimeyer found this to be the case in 14 per cent. of his 200 cases. Perhaps, as some have observed, when no pain exists the prognosis is more grave, and yet after all there are many healed latent ulcers. The pain is often relieved by rectal alimentation.

It is frequently localized and nearly always referred to the same spot in the epigastrium below the xiphoid cartilage. Pressure of the hand or clothing usually aggravates it, but in some cases, on the contrary, it gives relief. It may or may not be altered by a change of posture. One may say, as a general rule, that the effects of posture give no clue to the site of the ulcer; on the contrary, one may easily be misled through drawing false conclusions.

Tenderness.—Two spots of tenderness usually exist: (1) Epigastric and (2) dorsal. The most tender spot is usually in the midline of the epigastrium below the xiphoid cartilage, and sometimes a little to the left; or it may be located 2 or 3 cm. to the left of the spine between the tenth and twelfth dorsal vertebræ. It is an important diagnostic feature that the tenderness of the stomach corresponds to the seat of the pain after eating, and that the situation of this tenderness rarely varies during the whole course of the malady. The algometer of Boas, which has been used to determine the degree of sensitiveness, is supposed to indicate ulcer from epigastric sensitiveness to a pressure of 4 Kg. or less, and at the dorsal point to 5 Kg. Women are more sensitive than men.

The *cause of the pain* varies, it being apparently due sometimes to food, sometimes to the gastric movements, or to involvement of the peritoneum. Scars do not usually cause pain unless there are adhesions. According to Mansell Moullin, however, the gastric mucosa is non-sensitive, and hence the pain is not due to any exposed nerves in the ulcer, for there are none (at all events, probably no afferent sensory nerves), but rather to an inflammation

of the lymphatics near the ulcer and extension to the parietal peritoneum. The work of Pawlow would seem to contradict the statements of Moullin.

Lenander regarded the pain as due to adhesions or a subserous lymphangitis about the cœliac axis, while J. Mackenzie holds that it is viscerosensory reflex—a referred pain—and that the tenderness depends on muscular and cutaneous conditions rather than on the organic change in the stomach itself.

Vomiting is perhaps less constant than the pain, though Baltimore and Boston cases show the reverse. In Fenwick's 265 cases, 72 had vomiting, while pain was present in 100 per cent., and hæmatemesis, including melenæ, in 71 per cent. Cantlie found vomiting in 79 out of 85 of his cases in Montreal. Many other statistics give even higher figures. There may be merely nausea and flatulence without vomiting, or, again, the vomiting may appear only at intervals of a week or more. It has a very definite relation to pain, by which it is usually preceded. There is often no nausea, as one might naturally expect if the pain is the chief cause of the vomiting. It occurs sometimes immediately after taking food, but usually one, two, or three hours later, that is, during digestion and generally at its height. The pain is relieved by it. To some extent the onset of the vomiting depends upon the quality of the food, and occurs less often after taking fluids than after solids; it is especially easily induced by coarse, heavy foods. Perhaps sometimes the vomiting depends on infection from the mouth, through the swallowing of bacteria. In more or less acute ulcer there are no signs of motor insufficiency, and the vomitus consists merely of the food taken; in the older ulcers, where perhaps pyloric obstruction has already occurred and the stomach is dilated, the usual vomitus of motor insufficiency and gastrectasis may be present. The vomitus varies according to the food taken and the length of its sojourn in the stomach. As hyperacidity is usually present, one finds, as a rule, that digestion is quick and the albumins are well divided, although at other times the food is quite undigested, the vomitus acid, and the teeth are thereby put on edge.

Hæmatemesis.—Blood is frequently present in the vomitus, and occurs in more than 25 per cent. of all cases of gastric ulcer. In the statistics of the University College Hospital, the percentage was 84; at the Royal Victoria Hospital 60 per cent. had hemorrhage. Combined statistics collected from all sources, however, give hemorrhage in only 28 per cent. of ulcer cases. It is well to remember, in this connection, the many other causes of hæmatemesis that may exist, especially erosions of the mucosa. The oozing of blood may under such conditions be so great as to end fatally, and yet at autopsy show no signs of its origin. (Vide diagnosis of gastric ulcer for consideration of the various causes, *e. g.*, vascular disturbance, especially in mitral disease; cirrhosis of the liver; splenic anæmia, leukæmia, and in other blood dyscrasias; uræmia; various disorders of the mouth, throat, and lungs; tabes dorsalis, fraud, ingestion of wine, ice, fruit, coffee, fruit juices, etc.)

The source of the blood in gastric ulcer is usually arterial, the vessel becoming eroded during the development of the ulcer. At other times there may be simply a vein or capillary opened with slow oozing; or, again, there may be merely pore-like erosions of the kind referred to by Dieulafoy as "*exulceratio simplex*." Savariaud found in 54 cases that the hemorrhage was venous in 4, from the splenic artery in 17, from the coronary artery in 6,

from the pancreatico-duodenalis in 7, from the gastric arteries in 10, while in 10 others the source was undetermined.

One cannot always tell from the amount of blood what is the actual vascular source. The hemorrhage may be severe or slight. It may be fatal at once even before ejection from the stomach, the "hémorragie foudroyante," where a large artery is eroded; such cases form 3 to 5 per cent. of all fatal ulcers. Thanks to surgical intervention in recent years, the percentage is lessening constantly and progressively. Again, the blood may be vomited in large amount, or one may get, as is more common, especially in the more chronic cases, repeated hemorrhages of varying amounts. Hemorrhages are fatal in a large number of cases, according to Rodman in 40 per cent. and according to Mayo Robson in 64.2 per cent. Hemorrhage certainly forms about 20 per cent. of all the causes of death in ulcer.

Sometimes the bleeding is more latent, being not visible to the naked eye either in the vomitus or fæces, although minuter methods of detection reveal its presence. These "occult bleedings" have assumed a great importance in diagnosis in the last two years and much work has been done to enhance the value of this method.

Symptoms and Signs of Hæmatemesis.—The hemorrhage comes on usually after meals, probably owing to the action of digestion and the existence of distention, or, again, it may appear immediately subsequent to some effort or strain, such as from vomiting or defecation. Usually when there is a serious hemorrhage the patient vomits a large amount of bloody fluid, becomes pale, cold, and clammy, complains of dizziness, is often semi-conscious or more or less collapsed. Headache and thirst follow. The pulse is rapid and small, and there is dyspnoea or sighing, and often a warm feeling in the epigastrium is complained of. If recovery occurs, the reaction is apt to come on with fever.

The vomited blood may be bright or dark in color, bright if the vomiting be arterial, copious, and rapidly following upon escape of the blood from the vessel, because the corpuscles are comparatively unchanged. It is more usually dark red, sometimes brown, resembling coffee-grounds, the hæmoglobin having been changed to methæmoglobin by the hydrochloric acid, and few corpuscles are left unaltered. The vomitus, which may be copious, contains coagula, bits of food, is usually acid in reaction, and devoid of froth. There may be lesser repetitions of the hemorrhage, or after the one large one there may be spontaneous cessation. Hemorrhage may be divided into that from the acute and that from the chronic ulcer.

In the *acute ulcer* there may be a spontaneous, abrupt, and alarming onset, without previous warning and history. Much blood is lost quickly, and the patient either soon dies or the hemorrhage ceases of itself, with only slight repetitions and with anæmia; or there may be an abrupt onset, with short intermissions before one or more recurrences.

In *chronic ulcer*, on the other hand, the hemorrhage varies much. There is a history of chronic indigestion with vomiting for some time previously, and of persistent anæmia.

Moynihan divided these cases into four groups:

1. The hemorrhages which are latent and insignificant.
2. Those which are intermittent, infrequent, and moderate in amount. There is no danger in these of a fatal issue.
3. Those which have become worse after the symptoms have developed.

The bleeding may be copious and repeated, and therefore perilous, perhaps even fatal.

4. Those cases in which the hemorrhage is sudden, overwhelming, and fatal.

Sometimes the hemorrhage is repeated in spite of care, and according to Moynihan, is attributable to distention of the stomach and stretching of the ulcer.

Some of the blood reaches the intestines, and during the next day or two, the feces are black or brownish black, with a shiny surface. It does not, apparently, require a great deal of blood to produce a tarry stool, although Hosslin, quoted by Howard, states that 500 cc. are necessary. Sometimes the color is brighter according to the freshness of the blood, according to the amount, the rapidity of bleeding, and of its passing with the intestinal contents. The discoloration is due to the intimate mixing of the blood and chyme in the small intestines. Sometimes all the blood escapes per rectum and there is no hæmatemesis, which occurs when the bleeding is more gradual or the ulcer nearer the duodenum, with less tendency for vomiting.

"Occult Bleeding."—Strauss first drew attention to this valuable sign, and in 1902 Boas showed the diagnostic significance of the constant presence of occult blood in cancer of the stomach, and, following upon his observations, Kochmann, Schloss, Hartmann, Rossel, Schmilinsky, Rüttimeyer, Friedenwald, Steele, Cowie, Dewis, Einhorn, and others have recorded their experiences, all of which tend to show that the examination of gastric contents, vomitus, or feces for evidences of hæmatemesis and its underlying cause has considerable diagnostic value. It is a well-recognized fact that much blood may be present in the feces and yet go undetected, owing to alterations in its color and the nature of the diet administered. Schumm believes that stools may contain more than 5 per cent. of blood and yet give no evidence of its presence.

The examination for occult hemorrhages must, of course, be preceded by the exclusion of all other sources of blood, *e. g.*, bleeding from the gums or nose, injuries to the gastric or œsophageal mucous membrane from passing a tube (in which cases, however, the bright-red blood is obvious to the naked eye), general or systemic diseases, and diseases of the digestive organs. Of the general systemic diseases, one considers especially hæmophilia, purpura, scurvy, typhoid fever, arteriosclerosis, and tabes with gastric crises.

There are two other classes of diseases of the alimentary tract in which this symptom may appear and give rise to some confusion in the diagnosis.

1. Malignant tumors of the gastro-intestinal tract and of the pancreas or liver with ulceration, and also hemorrhagic pancreatitis. These give marked and often constant reactions.

2. Stenosing gastritis, alcoholic gastritis, polypi, intestinal parasites, tuberculosis, syphilitic or simple catarrhal ulceration of the bowel—also hemorrhoids, fistula, and fissure. All of these give constant and marked reactions. The ingestion of much raw or ill-cooked beef (200 gm.), blood sausage, etc., will likewise give positive reactions.

Extreme care in the preliminary dieting for these tests is unnecessary, for, as a matter of fact, there are but a few meats which yield positive reactions (*e. g.*, the above-mentioned raw, minced beef and rich beefsteak and blood sausages), and the quantity must be over 200 grams. Well-boiled or roasted meat does not interfere with the tests.

In suspected ulcer it is especially useful to examine the fæces after pain for the presence of blood. The following tests are of use: (1) Microscopic, for blood cells and pigment. (2) Spectroscopic. (3) Microchemical, *i. e.*, Teichmann's test. (4) Chemical. Where particles of material are found, resembling blood, one may merely use the microscope for the detection of blood cells and pigment, and may readily determine their presence in a few moments. Where doubt still exists, one may use the spectroscope with advantage.

Spectroscopic Test.—Dissolve a portion of the fæces in a small amount of water; add concentrated acetic acid; filter, and shake up the filtrate with ether. If blood exists, the ether becomes reddish brown and the spectrum shows the broad absorption bands of acid hæmatin in the red. It is less delicate than some of the chemical tests and more cumbersome.

The Microchemical Test of Teichmann.—This has been known for upward of fifty years. One mixes fæces and sodium chloride crystals on a glass slide and adds a coverslip. Then a drop of glacial acetic acid is allowed to trickle beneath; the slide is heated over the flame and the acetic acid steamed off; drop by drop more glacial acetic acid is added until the fluid becomes a distinct brown. It is well not to boil. Then it is evaporated in the air and examined for the crystals of hæmatin hydrochlorate (hæmin), *i. e.*, red-brown rhombic plates of small size. The test is good in dilutions of 1 to 20,000, but it is not always reliable, or so delicate as some of the chemical tests employed.

Chemical Tests.—1. Guaiac Resin. Many modifications of the original test as described by Schönbein, van Deen, His, Weber, and others have been made, and probably that of Cowie is most suitable. His directions are as follows: To one gram of stool, which has been softened, if necessary, by a few drops of water as possible, about 4 or 5 cc. of glacial acetic acid are added and intimately mixed. This is best accomplished by rubbing up in a glass mortar. To the above mixture add 30 cc. of ether and allow to extract for several minutes, after thoroughly shaking or mixing. To 1 or 2 cc. of the extract, which is perfectly clear and can be decanted off, add an equal amount of distilled water and shake thoroughly. To the resulting mixture add a few granules of powdered guaiac resin, an amount that will be held on the tip of a small knife blade; this is allowed to dissolve, and the tube is agitated thoroughly.

Thirty drops of old water-white chemically pure turpentine are now added and thoroughly mixed with the contents of the tube, which is set aside against the light or in front of a white surface.

If blood is present to the amount of 1 milligram to 1 gram of stool, a distinct light-blue color develops quickly in the upper half of the mixture, remains for a short time, and gradually disappears. Larger amounts of blood cause a more intense blue, but almost always a turbid or opaque blue, often the color of a turquoise. If the reaction is intense, it may be better seen if about 10 cc. of ether be added to the original extract. If smaller amounts of blood are present, the reaction may assume a smoky grass-green color. In such cases other tests, *e. g.*, benzidin should be tried as a control.

Dewis emphasizes the fact that "when turpentine is used as the oxidizing agent, it should be perfectly pure and clear, and before use should be exposed to the air in an open dish and in a dark place for at least a month;

afterward it should be kept in a bottle uncorked and only lightly protected with cotton."

The Aloin Test.—This was first described by Klunge and later by Rossel. A few grains of powdered aloin are placed in a clean test-tube and about 10 cc. of 75 per cent. alcohol are added. This yellow solution is used and the test performed as in the case of the guaiacum method. The resulting positive reaction gives a pinkish or darker red color, which in some instances is delayed for ten or fifteen minutes. Delayed reactions are doubtful, and the addition of chloroform hastens the test. The value of this reaction is about the same as that of the guaiac test.

The Benzidin Test.—This is probably the most suitable of all the chemical tests. First used by the Adlers in 1904, benzidin, one of the diphenyl group, has been recognized as the most delicate test hitherto employed for the detection of occult blood, giving positive results in blood dilutions of 0.001 per cent. The quality of the benzidin is of importance for its delicacy of reaction.

The test as modified by Schlesinger and Holst is as follows: A knife-pointful of benzidin is added to 2 cc. of glacial acetic acid in a clean test tube. A solution quickly ensues after shaking. In another test tube a tiny portion of fæces (size of a pea) is well mixed with 2 cc. of water, closed lightly and heated to boiling point. The reagent for use is made by mixing 10 or 15 drops of the benzidin solution with 2 or 3 cc. of a 3 per cent. solution of hydrogen peroxide. A few drops of the fæces mixture are added to this, and in the presence of blood a green or blue color appears in one or two minutes. If much blood be present, the blue color predominates.

Einhorn has simplified the test by preparing a *Benzidin paper*, made by moistening filter paper with a saturated solution of benzidin and glacial acetic acid and drying it. (One must avoid contact with the fingers, as a drop of perspiration causes a similar reaction.) His method is as follows: A piece of benzidin paper is immersed in the solution to be examined and a few drops of hydrogen peroxide are added. The paper is placed on a white porcelain dish and examined for the development of the blue color. This appears in positive reactions within one minute, or in great dilutions after a somewhat longer interval. Certain precautions must be observed in Einhorn's modification. Farina and boiled potato produce a reaction, so that in examination of the gastric contents the liquid parts alone must be used. Hydrochloric acid may likewise cause a reaction, but this is delayed beyond two minutes, so that one minute should be the maximal time limit for deciding upon a positive reaction.

The paper method is recommended as a valuable preliminary test, which, if doubtful, must be confirmed by the aloin ether extract method. For examination of the stools, one takes a small piece of the fæces, rubbing it up with 2 cc. of water, and immersing the benzidin paper. The hydrogen peroxide is then added and the color reaction observed. Certain limitations of this test must be noted: Some salts of iron, some organic fluids, *e. g.*, saliva and sweat, certain chlorophyll-containing vegetables if unboiled, all may give a positive reaction. The patient must therefore be dieted to some extent, preferably with lacto-farinaceous and egg foods. The action of the digestive juices will not prevent a reaction.

The Value of Examination for Occult Bleeding.—A negative result is so regarded only after at least three examinations have been made. The

value of this finding lies in its diagnostic, prognostic, and therapeutic aid. It is a premonitory sign of some lesion of the mucous membrane of the stomach. It is useful in the differentiation of neuroses, achylia, and anacidity, from certain organic lesions of the stomach. The intermittent presence of occult hemorrhages suggests gastric ulcer, whereas their constant presence is significant of carcinoma. Certainly, a persistent negative result renders the diagnosis of cancer dubious. In ulcer cases the occult bleedings are most constant before treatment is instituted and when pain and nausea are most marked.

Rütimeyer's statistics are of interest in this connection:

Ulcer Cases.—The gastric contents were examined in 35 cases (90 individual examinations); 13 gave positive results (37 per cent.). The feces were examined in 19 cases; 8 gave positive results (42 per cent.).

Cancer Cases.—The gastric contents were examined in 27 cases (78 individual examinations); 26 gave positive results (96 per cent.). The feces were examined in 19 cases (35 individual examinations); 15 gave positive results (79 per cent.). In the 27 cases where one or both examinations were made, positive results were obtained in all.

The comparative value of the test is shown by the fact that of 103 other gastric and intestinal diseases, only 13 per cent. gave a positive reaction. Friedenwald and Rosenthal confirm these observations. An examination made in 47 cases of chronic gastritis gave repeated negative results after many observations, as also in 15 cases of gastric atony, in 42 cases of simple hyperchlorhydria, and in 25 cases of nervous dyspepsia.

As regards prognosis, the test is of value in ulcer cases, inasmuch as marked or increasing intensity of reaction suggests an unhealed ulcer, and continuance in spite of medical treatment implies need of surgical aid. The therapeutic value of the test is obvious, and with proper treatment the occult bleedings diminish.

Objective Signs.—But little is to be gained from physical examination of the stomach in acute ulcer. Tenderness is the most reliable sign. A localized tender spot, often no larger than 2 to 3 cm. in diameter, may be present and may be felt through to the back on deeper pressure. It is found most often just below the xiphoid cartilage. Boas lays stress on tenderness near the twelfth vertebra on the left side. Very frequently the tenderness is not so localized, and a more diffuse soreness is felt in the epigastrium on palpation. No tumor is palpable in fresh ulcer. In older ulcers, where scarring has occurred to cause pyloric obstruction or deformities elsewhere in the stomach, the objective signs may be much more marked. Peristaltic movements, perhaps a dilated stomach, may be seen on inspection, while palpation may reveal a tumor of varying size, more or less movable, according to its situation and the presence of adhesions, situated usually at some part of the upper right quadrant of the abdomen. Reinhardt's cases showed 16 with tumor, in which a small gastric ulcer was found at autopsy. Moynihan in 100 gastro-enterostomies with ulcer found 10 with tumor, all of them benign ulcers. The palpable mass present is either from the thickened ulcer itself, from muscular hypertrophy or adhesions, or from exudate (Gerhardt).

Gastric Analysis.—This varies according to the type of the ulcer. In the acute forms the examination of gastric contents may show little that is normal. The hydrochloric acid may be normal in amount, the proteolytic

digestion unimpaired, and the motor power is usually good. At other times there may be hyperacidity or the reverse, and motor insufficiency may be evident. Blood is often present, and may be visible to the naked eye or found only by microscopic or chemical examination.

In the more chronic forms the findings will depend on the site of the scarring ulcer. If pyloric stenosis be present, there is usually hyperacidity with retention of food, good digestion of proteids, and little or no change in the carbohydrates. Sarcinae are usually found in such cases, and also yeast cells in abundance. Blood, too, either evident or occult, is common here. Lactic acid is present only where there is stagnation and hypochlorhydria. Rüttimeyer found motor insufficiency of the first or second degree in 42 per cent. of his cases.

Complications and Sequelæ.—The most important are: Perforative peritonitis, perigastritis (suppurative and adhesive), subphrenic abscess, fistula between the stomach and adjacent organs, abscess of the liver, chronic hepatitis, acute and chronic pancreatitis, constriction of the bile-duct with jaundice, cardiac stenosis, pyloric stenosis, deformities of the stomach with dilatation or hour-glass stomach, cancer of the stomach, general emphysema, parotitis (sometimes in cases with perforation, and sometimes after hemorrhage), tetany, hemorrhage, anemia, etc.

Perigastritis.—As a result of chronic ulcer, peritoneal reaction occurs in the vicinity, and may be infectious in origin. The resulting inflammation may be suppurative or merely adhesive. Adhesions occur to neighboring organs or the parietal peritoneum, and symptoms are thus produced which may give a clue to the diagnosis of the complicating condition; 5 per cent. of postmortem records show evidence of perigastric adhesions, and surgical experience indicates that these are extremely common. The adhesions form most commonly about the lesser curvature, in the midregion in front and behind, where the parts, too, are most at rest; also at the extreme ends of the organ, cardia and pylorus, described by the French writers, as pre-cardiac and prepyloric perigastritis. They are firm, well organized, and thick, and extend well beyond the limits of the ulcer and in extreme conditions even to the pelvis. The site of the ulcer determines to a large extent the location of the adhesions, so that with ulcers of the posterior wall the pancreas is involved, while with ulcers of the anterior wall and lesser curvature there are adhesions to the left lobe of the liver, the omentum, and the anterior parietal peritoneum. Sometimes even the spleen is adherent through new-formed bands. With pyloric ulcers the gall-bladder and liver may each be firmly bound to the stomach, and gastrectasis thus commonly ensues. Sometimes an hour-glass stomach is formed by means of these adhesions. Fenwick's statistics of 123 ulcer cases with adhesions show:

Organ.	No. of cases.	Per cent.
Pancreas alone	49	40
Liver alone	33	26.8
Both pancreas and liver	10	8.1
Colon	7	5.7
Liver and colon	4	3.2
Mesentery	3	2.4
Spleen	2	1.6
Three or more organs	15	12.2

Of 17 cases analyzed by Duplant, in 13 the adhesions were to the left of the median line, engaging the left lobe of the liver; in the other 4 they were

to the right in the pyloric zone, and of these, in 3 there were adhesions to the gall-bladder and bile-ducts with jaundice. These figures conflict with Brinton's statement that perigastritis is commonest at the pylorus and over the posterior wall.

The *symptoms* depend on the kind of perigastritis, whether suppurative or adhesive, its extent, degree, and location, as well as on the way the various neighboring viscera are implicated. It may be confused with gallstones or an angiocholecystitis, with secondary stenosis of the pylorus, or again with cancer, etc. Of course, many causes other than gastric ulcer may induce a perigastritis, *e. g.*, diseases of the gall-bladder, liver, pancreas, plastic and tuberculous peritonitis, hypertrophic stenosis of the pylorus, etc. The special features are pain, tenderness, and chronicity, without either cure or markedly progressive failure of health.

Signs of ulcer have usually been present for a long time previously, and to these are added exacerbations of the gastralgic pains, often radiating to the back or upward, to some extent relieved by rest, and increased on forcible straightening of the trunk. They have usually no relation to food. Some loss of flesh occurs, but there is no cachexia. Slight febrile attacks are common. Ingestion of food or fasting has no effect on the symptoms. Examination of the abdomen reveals perhaps nothing but tenderness, or there may be increased resistance and even a definite mass, which is subcostal, perhaps superficial.

With *pyloric perigastritis* the pains often radiate to the shoulder; insufflation of the stomach gives no aid to palpation because the adherent pylorus shows no changed position. The mass of inflammatory tissue is fixed and may be covered by the liver.

The differential points are briefly: A previous ulcer or one still unhealed, with a history of long standing and yet an absence of cachexia and metastases; perhaps gastrectasis; persistent pains more or less influenced by posture and sudden exertion; localized tenderness; occasional jaundice without involvement of the liver; periodical febrile attacks of short duration. Gastric analysis gives no distinctive results.

With *precardiac perigastritis* (which some regard as the commonest form) the signs vary greatly. There may have been signs of a persistent dyspepsia, with anorexia, constipation, gastric catarrh, and local tenderness, or, again, recurrent attacks of pain and fever, with increasing severity and frequency.

There is definite tenderness especially beneath the left costal margin, the right being free. Sometimes the pains radiate to the left breast, but not below the navel. There is no gastrectasis, but vomiting is frequent. Palpation sometimes reveals a plaque-like mass suggesting the possibility of cancer or tuberculous peritonitis.

Perforation.—This is one of the most serious complications of gastric ulcer, and occurs in a varying percentage of cases. Some authors place it at 15 per cent. of all ulcer cases, others at 6.5 per cent., and others again much less (Greenough and Joslin, 3.2 per cent.). Cantlie found it in 10 per cent. of the Royal Victoria Hospital cases. In Lebert's clinical statistics of 252 cases, the percentage was 3.5, while Fenwick in his 265 cases found it in 5.5 per cent. Clinically, in 1336 cases there were 45 perforations, *i. e.*, 3.4 per cent. In 1615 autopsy cases there were 336 perforations, *i. e.*, 22 per cent.

Perforation would seem to be more common and to occur at an earlier age

in women than in men. In 127 perforations from various sources, 99 were in women, *i. e.*, 76.4 per cent. It may occur several times in the same person, as in a case reported by G. R. Anderson, and Mr. Caird of Edinburgh had a similar experience, two perforations occurring in the same patient within fourteen months. According to Robson and Moynihan, multiple perforations are more common than is generally supposed (20 per cent.). A remarkable case is reported by Keays, in which two perforations, one anterior and one posterior, seem to have occurred simultaneously, and were followed by a fatal result and autopsy.

Perforation occurs from the deeper extension of the ulcer, and the "depth of the extension seems to depend on the size of the area and the rapidity of the necrosis, determining the extent to which secondary inflammatory thickening of the subjacent peritoneum could occur, and also on the nature of the infecting germ" (Brooks). It also depends on the location in relation to solid viscera, and an ulcer is more apt to perforate when adjacent to a hollow cavity. For this reason perforations are more common on the anterior wall, where there is great mobility in the part and less tendency to the formation of adhesions. The *results* depend upon two factors: First, the site, and secondly, the rapidity of the ulcerative process. As regards the *site*, Brinton's statistics give 70 per cent. on the anterior wall, 21 per cent. on the lesser curvature, and 9 per cent. on the posterior wall. If the perforation occurs on the anterior wall, there is usually extravasation into the free peritoneal cavity.

If the lesser curvature be its location, the contents appear in the lesser peritoneum; while if again the posterior duodenal wall be perforated, it permits extravasation into the cellular tissue behind, or else along the ascending colon or about the kidney.

Pariser and Lindner say that of 200 cases of ulcer, 190 will be on the posterior wall and only 4 will perforate, while of the other 10, $8\frac{1}{2}$ will perforate.

According to Moynihan, perforations may be *acute*, *subacute*, or *chronic*. In the *acute* cases, which are the severer ones, the symptoms are sudden, the leak is opened to the peritoneum, and signs are general from the beginning; in these the perforation is usually on the anterior wall. The *subacute* cases are those in which the stomach is empty at the time and the perforation small. Perhaps the omentum blocks it, or an early adhesion forms with a small leak and a limited peritonitis or local abscess. These cases are often preceded for some days by symptoms of pain or tenderness, slight or severe, and perhaps only felt on movement, laughing, etc. Phlegmonous gastritis is very rarely the sequel of ulcer (v. Mintz, Merkel, Glaser, etc.).

The *chronic* cases are those in which there are protective adhesions which have formed before actual perforation, and the symptoms are ephemeral or those of abscess formation elsewhere. Partial or general peritonitis may ensue and adhesions form between the stomach and adjacent organs, especially the pancreas, liver, and the omentum; we may get a *subphrenic abscess*. When partial the peritonitis is usually confined to the epigastrium, and is, as a rule, purulent. If posterior, pus may collect in the lesser omentum. Adhesions may close the foramen of Winslow and temporarily prevent the spread into the general peritoneal cavity.

General peritonitis occurs chiefly when the perforation takes place rapidly before adhesions form. The extent depends upon the time from onset, for

the transverse colon and the mesocolon act as temporary barriers for a few hours before extension occurs into the lower half of the peritoneal cavity (Berg).

Course of Perforated Ulcer.—Sometimes, though rarely, an acute perforative general peritonitis may heal spontaneously. This is extremely rare in the acute cases. Some subacute and chronic cases recover, although usually with conditions which lead to more or less chronic invalidism. Statistics show that 95 per cent. of cases untreated surgically die; that 25 per cent. of cases operated on within twelve hours of rupture die; that 63 per cent. of cases operated on within twelve to twenty-four hours of rupture die; and 95 per cent. of cases operated on twenty-four hours after rupture die. *Extension* may occur in almost any direction. By contiguity, directly or indirectly, adhesions may form with adjacent organs or tissues and the results vary; sometimes they are serious, in which case the symptoms are associated with the disease of the part newly affected, or else no serious symptoms develop and healing occurs. *Liver and pancreas:* Perforation into the liver or pancreas may occur with abscess or chronic inflammation in either organ. There may be obstructive jaundice with pressure on or stricture of the ducts and a *catarrhal cholangitis*. Subphrenic abscess may occur, and was due to ulcer in 20 per cent. of 179 cases (Maydl). The *portal vein* is sometimes invaded, and pylephlebitis follows. The *gall-bladder* may be involved and stones may fall into the stomach through the fistulous opening. Sometimes the *spleen* is involved, and we may find a perisplenitis.

If the perforation penetrates the *intestines*, there may be a severe diarrhoea and bloody or purulent faeces or faecal vomiting. A gastrocolic fistula will form. (Murchison found 10 cases of this in 33 perforations due to ulcer.) Bertholdstein, of Nuremberg, records a case in a girl aged twenty-six years, in whom perforation occurred simultaneously into the colon and the pelvis of the kidney, and there were quantities of pus in the stools and urine; the patient ultimately made a good recovery.

Rarely perforations occur into the *abdominal wall* with an external fistula. These cases have been studied by Murchison (12 out of 25 cases) and by Middeldorpf and Kronheimer. Sometimes a general subcutaneous emphysema may follow such a complication. The *pleura* is not infrequently involved, with a resulting pyopneumothorax, or the lung may rarely be invaded and contain stomach contents. Tillmann collected 12 such cases, all fatal. The *pericardium* is known to be invaded sometimes, and even the left ventricle of the *heart*. Of the 28 cases reported by Pick, in which the *diaphragm* was perforated, 10 had invaded the *pericardium*.

In the more chronic cases sometimes the perforation occurs at a place far distant from the original ulcer, by dissecting and pointing, as in one instance where a pelvic abscess formed and was drained through the vagina some weeks after the perforation of the ulcer had occurred.

Perforation of a gastric ulcer sets up a general peritonitis, more commonly than does a duodenal ulcer, perhaps because the duodenal contents are more sterile, or because there are more chances of adhesions. The same rule applies to pyloric ulcers as compared with ulcers elsewhere in the stomach.

The Symptoms and Signs of Perforation.—The early signs are the most characteristic, hence the importance of seeing cases early, but there are no pathognomonic symptoms previous to perforation. The onset is usually sudden; indeed, the sudden pain or other distress may be the only sign of the

ulcer. English found no history of stomach trouble for some time before perforation in 11 of 50 perforation cases. The patient need not be overcome and incapacitated at once; sometimes he is able to walk into the hospital with such a perforation. In one case under Seymour Taylor's charge, there was a single bloody stool as the only sign of perforation, without pain or abdominal signs of any kind, and a fatal result ensued with no other local development. A patient of Shuter's, of London, came running to his office on account of abdominal pain, and showed no signs of collapse for several days after the perforation had evidently taken place. It commonly follows some strain, such as vomiting, bodily exercise, etc., or trauma, or a hearty meal.

Pain is the earliest symptom, usually referred to the epigastrium, spreading soon to the right and left, but not leaving its original site. There is no pain or difficulty during micturition; dyspnoea soon follows, and deep breathing causes pain about the diaphragm, so that these cases are sometimes mistaken for acute pleurisy. Collapse and prostration supervene. In some cases remarkably little prostration is observed, a fact which is apt to mislead, especially as the general signs may greatly improve after a few hours before graver symptoms develop. The degree of collapse depends in large measure on the amount and extent of the extravasation. The face is anxious, often pinched, and there is apt to be great restlessness. The pulse is usually accelerated, although sometimes it is normal, and the temperature may be but little altered immediately after perforation has occurred. Usually there is slight fever. The abdomen may be flat and tense, sometimes it is distended; the muscles are rigid and there is diminished mobility, two signs of greatest significance. Tenderness is chiefly in the upper abdominal zone, mostly about the epigastrium and hypochondrium. Eighteen out of a series of 49 cases were diagnosed as appendicitis. Percussion may or may not give dullness here, and the liver dullness need not be obliterated, although this depends in great measure on the escape of gas into the surrounding parts. The sign of obliteration of the liver dullness is unreliable. Cases of uncomplicated gastric ulcer without distention of the abdomen have been published, in which liver dullness was absent and in which laparotomy had been performed and no perforation found. In 43 perforation cases, English found the area of liver dullness normal in 11, absent in 12, diminished in 20. Even when absence of liver dullness exists as far back as the midaxillary line, it may imply no abnormality. Nor, again, is it true that well-marked stomach tympany in the left hypochondrium excludes the possibility of perforation. A valvular perforative opening may exist allowing distention. On auscultation, a friction rub can often be heard over the diaphragm, and sometimes the gurgling of fluid through the perforation.

Pseudoperforation is not uncommon, *i. e.*, the signs and symptoms of perforation without other anatomical lesion than mere ulcer, as in a case recorded by Manges, in which a woman, aged twenty-seven years, after previous signs of ulcer (hematemesis, etc.), suddenly had pain and tenderness in the epigastrium, with a temperature of 102° , pulse 120 to 140, and marked rigidity. Operation was performed, but no perforation and no peritonitis were found, simply the uncomplicated gastric ulcer. Leukocytosis varies in this as it does in the perforation following typhoid fever. It is commonly taught that vomiting never occurs after perforation, but this cannot be relied upon, as experience teaches the fallacy of too strict an adherence to this general rule.

We are told that attention should be paid to the base of the thorax on the left side, where one often obtains restricted movement, localized pain, and tenderness, with dulness in the lower axillary region and other signs of fluid (Osler). Fluid may collect beneath the diaphragm about the anterior and external surface of the spleen, or in the lesser peritoneum and later on the usual signs of general peritonitis may develop.

Cancer Developing upon Ulcer.—Cruveilhier first suggested this possibility in 1835, although he believed a cancerous diathesis essential to its development, and Rokitansky, in 1839, noted its occurrence and the possibility of its frequency.

Dittrich, in 1848, analyzed 166 cases of cancer of the stomach, and found 8 in which it seemed to have developed on an old cicatrix, and 2 where the very edge of the ulcer was the starting point of the malignant disease. The first microscopic examination of such a condition, however, was made by Waldeyer, in 1867, who regarded it as the result of an active process in still fresh ulcers. Later on Lebert found it in 9 out of 100 cases, and finally Hanser and Zenker, by careful histological observations, established the relationship of cancer and ulcer beyond all doubt in 1883. Curiously enough, Kollmar never saw a case of the kind in twenty years' experience at Tübingen.

More and more has the idea gained ground that ulcer is one of the commonest predisposing causes of gastric cancer, until recently Graham, working at the Mayos' clinic, suggested after an analysis of the cases there that probably 61 per cent. of all their gastric cancers had a previous history of ulcer.

That the cancer may be frequently associated with ulcer is further suggested by the frequency with which each selects the pylorus and lesser curvature, and from the fact that cancers elsewhere so frequently originate in scars, all the more so in the stomach, where the cicatrices are liable to constant or intermittent irritation. The cases are usually prepyloric or juxtapyloric.

The malignant tumor usually arises from the edge of the ulcer, where the glandular loops have undergone changes. The base is smooth and hard, and shows less granulations than the ordinary chronic ulcer. The edges are thickened and rounded, and the mucosa itself shows further ulcerations and friability. There is a tendency to adhesions of the base with neighboring viscera, *e. g.*, pancreas, and in this manner, too, perforations often occur. The remainder of the stomach may be normal.

The Symptoms and Diagnosis of Cancer Forming on a Previous Ulcer.—The transition period between the signs of the original ulcer and the onset of the cancer varies greatly. Sometimes the duration of symptoms is short, so short as to make the sequence dubious, and yet full pathological proof is available. At other times the earliest symptoms of ulcer may have existed so long previously as almost to have been forgotten. Long periods of latency are common. The transition is not always easy to tell, and conclusions are chiefly based upon the history of the course of the malady.

In patients known to have had ulcer previously, malignant development is suspected where loss of weight persists with a cachexia greater than a mere ulcer would justify. In such cases, too, there is often more pyrosis, although perhaps this is less severe; there is more constant pain, although perhaps even less intense, and its character alters to a more dull, sickening ache. Tenderness is more diffuse. There is more nausea and vomiting;

at all events, the vomiting is more regular, and even if a longer time intervenes between the attacks, it is more copious and rancid, readily excited by liquid food. Blood appears more frequently in the vomitus, and in small amounts, and occult blood is more constant in the feces. The appetite usually becomes less, the patient is more languid, nervous, and weak, and is markedly anæmic. The gastric analysis is not always characteristic of cancer, and then only late in the disease, and often hyperacidity is present to the end.

Mechanical and medical treatment are without avail, and if, with proper ulcer treatment, we get increase of the symptoms, especially of emaciation, pain, vomiting, anæmia, and weakness, cancer may be suspected. Sometimes the ulcer symptoms predominate to the end, and perforation may occur. The diagnosis is easier, when a long time exists between the ulcer and the newly developing cancer, and especially if combined with gastrectasis and the above symptom complex.

Parotitis.—Parotitis is not uncommon. S. Paget and Hone mentioned several cases in 1897, and English reported 5 instances among 50 cases with perforation at St. George's Hospital. In 2 of these latter the disease was bilateral. Sometimes suppuration occurs. The condition probably arises in a similar manner as in other diseases of the abdominal and pelvic viscera. Rolleston and others, while dubious as to its causation, do not regard the complication as one arising from infection of Stenson's duct.

Tetany.—Tetany, while commonly associated with old scarred ulcers with dilatation, sometimes occurs without any such complication. Moynihan had 5 cases. There are mild and severe grades, the former with mere cramps and paræsthesias at times, the more severe forms resulting often fatally. Albu reported 31 deaths out of 40 cases. Any source of irritation may induce an attack, and not infrequently the mere lavage of the stomach which so often relieves the symptoms may at other times precipitate an attack. The tetany is supposed to arise from some auto-intoxication due to absorption of decomposed gastric contents.

Diaceturia.—Finkelstein, Edsall, and others have recently drawn attention to the frequency of acidosis in certain gastro-intestinal affections, especially in children, and Rolleston has observed the common occurrence of diacetic acid in the urine of patients suffering from gastric ulcer (33 out of 38 cases). The toxic symptoms appeared chiefly with restricted feeding, and disappeared often after ingestion of glucose. Cases with marked and prolonged vomiting almost invariably had diaceturia.

Pyloric Stenosis with Dilatation.—(Vide article on Gastrectasis and Motor Insufficiency.) Dilatation is fairly common and is associated either with pyloric stenosis from a scar, ulcer, or perigastric adhesion as direct causes, or with contraction or spasm of the pylorus and mechanical irritation as indirect causes.

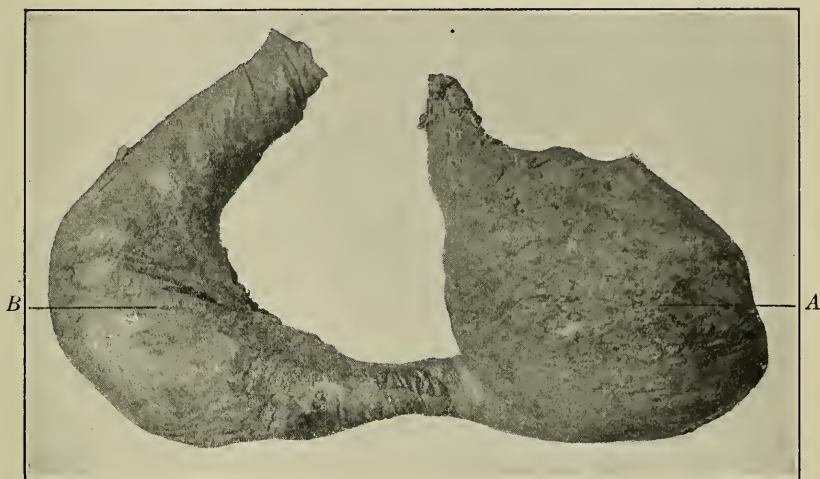
Cardiac Stenosis.—Cardiac stenosis from cicatrized ulcers at the cardiac end of the stomach may occur, as also diverticula at various portions from similar causes, or through traction of perigastric adhesions.

Hour-glass Contraction of the Stomach.—*Sanduhrmagen, L'Estomac Biloculaire.* This condition has been specially studied by Mayo Robson, Moynihan, Fenwick, and R. Williams, and is much commoner than was supposed. It consists of a contraction anywhere between the cardia and the pylorus dividing the stomach into two or more portions; sometimes it is trilocular. Moynihan has described a case with four sacs. As a rule,

the greater curvature is tucked upward, while the greatest thickness is at the cardiac portion.

Etiology of Hour-glass Stomach.—The condition is rarely a congenital one. Moynihan denies any such origin, and deals with individual instances which he claims as being inaccurately described as congenital, regarding it as almost always dependent on some organic disease from one of three causes—ulcer, perigastric adhesions, or carcinoma. Nevertheless, in one of the specimens in the McGill Museum removed from an infant, the condition is so marked as to leave no other conclusion than that it was, at all events in that instance, congenital. Meckel has the same view of its possible origin. The direct and most common cause is an old ulcer with cicatrix or adhesions of the stomach to the wall or other parts, or both, or the formation of independent

FIG. 2



Hour-glass contraction of the stomach: A, cardiac portion; B, pyloric portion.

bands of fibrous tissue. Among 48 cases reported by Robson and Moynihan in November, 1906, 41 were due to ulcer. Tuberculous peritonitis may cause bands of fibrous tissue to form about the stomach and produce identical results.

The symptoms are at times quite indefinite, and at other times appear to be quite characteristic. They apparently depend on the original cause at first, but later the effect may be diagnosed provided one exercises sufficient care and patience. As has already been pointed out by others, the main symptoms are pain after meals, vomiting of stomach contents and sometimes also of blood, emaciation, and the presence of a tumor due to cicatrization of old ulcers, with an absence of secondary nodes and of ascites. The condition is naturally associated with symptoms for many years, and the patients suffer much. For a time the symptoms seem to simulate stenosis of the pylorus, the presence of pathognomonic features depending largely on the site of the contraction. This, as a rule, lies four inches from the

pylorus, and therefore simulates pyloric obstruction with dilatation, all the more so as hyperacidity usually co-exists and the secondary cavity is small. Moynihan gives the following sets of symptoms, the result of the experience of various authors: *First*, in lavage of the stomach, all the fluid fails to return, some, which is in the secondary pouch, being apparently lost (Woelfer's first sign); *second*, after lavage has apparently thoroughly cleaned the stomach, more contents reappear, probably foul and evil-smelling, the washings thus being again dirty (Woelfer's second sign), or the same may be found on withdrawing the tube after washing the stomach clean and then passing it again; *third*, the sign of paradoxical dilatation (Jaworski); this consists in the succussion splash on palpation after apparent removal of all the gastric contents, a sign which is of course present because only the cardiac sac is emptied; *fourth*, after percussing out the stomach and then after distending it, a change in the position of the distention tumor is found at times. First the proximal pouch is distended and then the distal, and the notch is often visible between the two tumors when both parts are dilated (von Eiselsberg); bubbling and sizzling are distinguished at a point distinct from the pylorus on dilatation (von Eiselsberg), and patients are sometimes themselves conscious of this and of food passing from one pouch to the other. This may be detected by the stethoscope.

Diagnosis.—Numerous conditions are mistaken for gastric ulcer, inasmuch as the symptoms are at times very indefinite. The essential features in the diagnosis are: the general condition, the history, the sex; pain after taking food; tenderness in the epigastrium, usually over the site of the pain; vomiting, usually following the pain; hæmatemesis; dyspepsia in intervals between the attacks, and the presence of occult blood in the stools. The diagnosis of the *seat of the ulcer* is usually impossible. The ordinary evidences mentioned by many writers are now known to be quite unreliable. Again there may be several ulcers present at the same time, a fact which will still more confuse the efforts to locate the site. In typical pyloric cases, however, or in duodenal ulcers, it is not so difficult. The tenderness is usually to the right and pylorospasm may be observed. The pain, although variable in time of onset, may come on at least one hour after food, and in ulcers of long duration there may be gastrectasis. Tenderness and a tumor together suggest an ulcer of the anterior wall, but all such signs are of doubtful value. Hyperacidity is scarcely in itself sufficiently common to warrant the systematic use of the stomach tube for diagnosis in these instances. Certainly, this is contra-indicated when there has been recent hemorrhage, when there is any suspicion of an ulcer at the cardia, or, lastly, when little doubt exists as to the presence of a gastric ulcer. Although many assert that there is little danger in the careful introduction of the tube, one cannot deny that there is always some risk of precipitating a perforation, of starting up a hemorrhage, or of inducing severe vomiting, which in itself is not always harmless. Moreover, the frequent presence of hyperacidity in so many other conditions, nervous and organic, renders its finding, after all, of no very great importance, while, on the other hand, its frequent absence in gastric ulcer makes it still more reasonable to avoid the use of the stomach tube when no information other than this is available.

In the *differential diagnosis* one must consider simple gastralgia, hyperæsthesia, simple hyperacidity, hysteria, gastritis, pregnancy with hyperemesis, uræmia, cancer, gallstones and stones in the kidney, aneurism of the

abdominal aorta, and even spinal disease; also hemorrhagic erosions with exfoliation.

Gastralgia.—In gastralgia there is paroxysmal pain, not directly excited by food, nor related to or regularly affected by its ingestion. Pressure often relieves the pain; there are no tender points. If there be vomiting (and usually this is inconstant and irregular in onset), it does not necessarily give relief to the pain. Digestion itself is normal, and milk or other bland diets are of little use in the treatment. Usually there are eructations of gas without odor. There is no altered taste in the mouth, the appetite is capricious, and constipation is marked. Mucous colitis may be and often is superadded. Epigastric pulsation is common. Hæmatemesis is absent, as also occult blood.

Simple Hyperchlorhydria.—In this condition there is rarely vomiting or hæmatemesis, no occult hemorrhages, very little tenderness, if any, nor are there dorsal tender points. The pain appears from one to three hours after meals, sometimes at night, especially if there be hypersecretion. There are definite nervous causes, as a rule, for the condition, and there is great relief with proper diet, especially albuminous food, combined with alkaline treatment. In fact, if the hyperacidity be unyielding to treatment and the pain comes on rather soon after meals and with frequent relapses without known cause, one may suspect the hyperchlorhydria to be due to an underlying ulcer; so usual is it for proper treatment to be followed by successful results in the simpler condition.

The laboratory findings can scarcely be considered worthless in this differential diagnosis, but they are certainly often misleading. It is true that much hydrochloric acid points more to the possibility of ulcer, but one cannot lay too much stress upon such a finding.

Simple Hyperæsthesia.—In this there is usually pain after meals, with vomiting. The pain, however, appears at once after meals, and is irrespective of the kind of food or of its temperature. There is less localization of the tender spots; the vomiting has more variation, and may be always after meals, or irregular, and may give no relief. Only a portion of the food is ejected and the nutrition may remain good. The hydrochloric acid is not much in excess, and the rest cure is of little avail unless through suggestion. Occult blood is absent.

Hysteria with Vomiting.—In this there is no severe pain, as a rule; vomiting comes on at once after meals, part of the food only is ejected, especially liquids. There is no local tenderness; the nutrition remains good; dietetic treatment is of no avail and there are stigmata of hysteria elsewhere.

Gastritis.—A cause is present; the pain is more diffuse, less severe, and not increased much on pressure. The tongue is foul and coated. The vomitus contains much mucus, often organic acids, and there is diminution in the amount of free hydrochloric acid. If blood be present it is rarely large in quantity.

Pregnancy with Hyperemesis.—Here the history of the case and the local findings form the best means of making a diagnosis. Pain is, of course, almost always absent, and there are no tender points and no bleedings, unless a small gastric vessel be eroded from the strain of vomiting.

Uræmia.—Gastralgia often accompanies uræmic symptoms, but there are other signs present, and the urine will probably be characteristic.

Cancer.—In cancer, debility and emaciation often precede the other signs; the pain is more constant and more independent of food, is often nocturnal,

although it is true it may at times be quite absent, and there may be simply a discomfort. The breath is foul and there is anorexia, as a rule; vomiting, which is common, gives less relief than in ulcer. The vomitus is rather that of motor insufficiency, and shows little or no hydrochloric acid, but usually some lactic acid, and other evidences of fermentation in the odor and in the presence of the ill-digested food and the Boas-Oppler bacilli. Any hæmatemesis that occurs is more of the character of coffee-ground vomitus. Occult bleedings are more continuously found than in ulcer. The case is somewhat different where ulcer exists as a pyloric tumor with dilatation of the stomach, or where a cancerous growth has been engrafted upon a previous ulcer. In the first place, cancer limited to the pylorus is rare, and later, when the tumor develops, there are other signs. If it be limited to the pylorus, the onset is obstructive, with vomiting, cramps, emaciation, in a more elderly person; the course is progressive, and dietetic treatment affords no relief. If first seen when dilatation is really pronounced, the diagnosis must be made chiefly from the history and an examination of the gastric contents. Free hydrochloric acid is absent in 90 per cent. of these cases and lactic acid is evident. With ulcer there is usually hyperchlorhydria or a normal amount of hydrochloric acid. The history of ulcer causing dilatation is of years' duration, because the condition is one of gradual development. The appetite improves with lavage, as also does the general condition. Where there is a cancer engrafted upon an old ulcer, there is increase in the pain, especially if there be some emaciation and weakness; the hydrochloric acid is gradually lessened; and the tumor increasing in size assumes a more nodular character.

Hemorrhagic erosion with exfoliation of bits of mucous membrane; the lavage water often shows portions of the mucosa. Here, as in ulcer, there is a burning, often agonizing pain, and not infrequently there is blood in the vomitus. The differences from ulcer are not always easy to determine, but the crucial test is in the findings from the lavage of the empty stomach, in which bits of mucous membrane are present in the water removed. The pain comes on usually some hours after meals, and often irrespective of their character (whether solid or liquid), and at first the ingestion of the food may give relief for a time, the reverse of what usually takes place in ulcer. The use of silver nitrate is often very beneficial in these erosions.

Hemorrhagic Oozings from the Mucous Membrane.—In addition to these cases, however, there are others in which considerable hemorrhage follows minute erosions, so that ulcer is diagnosed, and yet nothing can be found anatomically. There is no means of differentiating such conditions with our present methods, and no definite clinical picture exists which can be associated with this condition to the exclusion of gastric ulcer. Although it is said that hemorrhagic oozings are more common in women; that pain occurs after food as in ulcer, but is more diffuse; that vomiting is more rare, and often without hæmatemesis as well as with it; that the blood is bright red and the hemorrhages may recur for years, often at long intervals; and that there are fewer tender points; yet there is scarcely enough of a symptom complex to warrant assurance in the diagnosis. Hale White has written at some length on the subject, and draws special attention to the fact that such oozings are neither vicarious nor hysterical, nor the early erosions of gastric ulcer. He regards the cause as obscure, the prognosis good, and holds that most patients recover.

In ulcer, too, there may be little or no vomiting and little or no tenderness, and the pain, also, may be diffuse, while even the presence of fragments of mucous membrane is possible in so many conditions that its occurrence scarcely calls for comment. The writer has seen a large portion of mucosa come away in the washings from the stomach of a healthy physician, who was experimenting on the chemistry of his own digestion. The microscopic appearances were those of a healthy mucous membrane.

Biliary Colic.—Gallstones: The differential diagnosis is often extremely difficult, and in a number of cases quite impossible for the time being. This is especially the case where the ulcer lies at the pylorus or in the duodenum. There may be here persistent pain, as in ulcer, especially after the taking of food, and with it vomiting; the vomiting may give relief; there may be tender points, and upon careful treatment even the symptoms may for the time disappear, and all this without jaundice, bile in the urine, or other signs of gallstones. As a rule, however, there is sudden, colicky pain at any time, of great severity and subsiding suddenly, tenderness in the right hypochondrium, slight fever, and perhaps sweating. Retching and vomiting occur with the pain and give no relief, pain continuing long after the stomach is empty. There are intervals of health and normal digestion. The co-existing gastralgia may be paroxysmal, and there is often hyperacidity. Not infrequently the sclerotics are subicteroid, and a trace of bile may be found in the urine.

Arteriosclerosis.—This occurs usually in old people, and is sometimes accompanied by abdominal pain. There is no local tenderness, the digestion is good, and in spite of all treatment the pain persists. In one case of gastric ulcer with hemorrhage, in an elderly man, a diagnosis was made of aneurism of the aorta, perforating the œsophagus, the diagnosis being suggested from the absence of gastric symptoms, the history, and the marked arterial signs.

Spinal Disease.—Rarely spinal disease has been mistaken for gastric ulcer, and plaster jackets have been applied for the treatment of gastric ulcer.

The gastric crises of tabes, the pains of myelitis, the pressure of a movable kidney, may all simulate gastric ulcer; likewise the pains of lead poisoning. The individual features and the routine examination will in each case clear up the doubts in the diagnosis.

Duodenal Ulcer.—This deserves special mention in view of the abundance of recent literature devoted to the subject and on account of the special symptoms presented by ulcers of this location. Pathologically these ulcers are found in 0.297 per cent. of all autopsies—*i. e.*, in 232 cases out of 78,000 autopsies (combined statistics).

The proportion of duodenal to gastric ulcers, which according to statistics is ever increasing because of the surgical findings, is perhaps 1 to 1. Former statistics give 6.6 per cent. of all peptic ulcers, but this is certainly far too low an estimate, and surgical experience bears this out each year more and more. In 1906 Mayo had found the proportion 2 in 5. Of his last 200 cases operated upon (1905 to 1907), 98 were duodenal and 87 gastric, while 15 were independent of each viscus. He drew attention to the canal of Jónnesco (the terminal three-fourths of the pyloric end) as being usually free in cancer, while the lesser curvature was often bridged over, like a saddle, near the pylorus, by the ulcer.

As regards age, the majority occur between the ages of thirty and forty years. The following analysis of 330 cases shows the age ratios:

Years.	Cases.
0 to 10	42
10 to 20	28
20 to 30	60
30 to 40	66
40 to 50	54
50 to 60	47
60 to 80	30
80 to 95	3

Oppenheimer found 15 duodenal ulcers in infants, in which *mekæna neonatorum* was the prominent symptom, and Chvostek records 5 out of 87 autopsies on children under ten years of age. Henoch, T. D. Lister, Spiegelberg, and others also record cases in infants.

FIG. 3



Duodenal ulcer.

As regards sex, of 917 cases, 79 per cent. were in males and 21 per cent. in females, although Mayo and Moynihan found a somewhat less disproportion between the sexes. Seymour Taylor reports 72 out of 100 cases in males. MacCallum and Tull have analyzed 23 cases occurring in the Royal Victoria Hospital, Montreal; 15 were in males.

As regards site, of 454 cases, 91 per cent. were in the first portion, 7 per cent. in the second portion, and only 2 per cent. in the third and fourth portions. Practically all of those in the first portion extend to within three-quarters of an inch of the pyloric sphincter, and the deepest portion is

usually just below the pylorus, where the acid chyme most affects the intestinal mucous membrane. In 6 out of 10 cases in MacCallum's series the ulcer was on the posterior wall; in the other 4 it was situated anteriorly.

Number.—Collin found one ulcer alone in 80 per cent., two ulcers in 11 per cent., three to four ulcers in 4 per cent. When multiple they are usually crowded together in the first portion. More rarely they exist simultaneously in different parts of the duodenum. MacCallum's statistics record only 3 out of 23 as being multiple. Moynihan, in 1902, drew attention to the co-existence of gastric and duodenal ulcers. In 44 cases of duodenal ulcer, one-half had gastric ulcer as well, while Mayo found even a greater proportion in his own experience.

The Symptoms of Duodenal Ulcer.—While, as in gastric ulcer, many cases run a latent course (91 out of 151 of Perry and Shaw's cases), yet, as a rule, pain is the prominent symptom, and is present in 80 per cent. of all cases. Its character varies within wide limits, and though sometimes slight and more like a mere burning in the midline or to the right of the costal margin, at other times it is severe and so acute as to incapacitate the patient completely. In several recent cases the pain resembled severe biliary colic, for which, indeed, it is often mistaken.

In one instance the pain occurred at long intervals (weeks or months), but when present was extremely severe, the patient being doubled up and in a cold perspiration. It was only after several months of such intermittent suffering that blood was finally discovered in the stools, and the subsequent operation confirmed the suspicions. Throughout there had been no vomiting. Remissions of the pain formed one of the features in our own statistics, and in 5 cases lasted from one to five months. Radiation of the pain is by no means the rule. It is usually local only, although at times it radiates to the breast, shoulder, or back. While usually related to the ingestion of food, in that it comes on from one to four hours after, it is not necessarily so. It is at times relieved by food, drink, lavage, or alkalies.

Under certain conditions any relation to meals may be quite absent, especially if peritonitis exists with or without perforation, or if there be no hyperacidity or motor insufficiency. In these cases the pain is due either to the local peritonitis, to spasm, the action of acids, or, again, to gaseous distention.

Tenderness, if present, is perhaps more frequently to the right of the median line, although quite often only in the epigastrium. It is at times diffused and not localized. It is rarely posterior.

Vomiting is less common than pain, being present in less than 20 per cent. of the cases. In MacCallum's series, however, it was present in 20 out of 23 cases, and frequently followed the attacks of pain. It is irregular, delayed, often acid, and the amount varies according to the amount of obstruction and of hypersecretion.

Acidity is usually increased, and hyperchlorhydria has been frequently found. The appetite remains good and constipation is the rule.

Hemorrhage from the stomach or bowels is common. Oppenheimer found melæna in 50 per cent. The diagnosis is made more commonly from the association of the local tenderness and the melæna, or even from the tarry stools in the absence of pain than from any other signs. Sometimes hæmatemesis and melæna occur together, and there may be so much hemorrhage as to produce a fatal result. It is severe in at least

one-third of the cases and frequently even in these it is the only sign present except for the secondary results, such as pallor, relapse, reactionary fever, etc., or, again, these signs may precede the appearance of the blood by some hours or days. The hemorrhage of duodenal ulcers is more difficult to check and usually of longer duration than that of gastric ulcer. In the 23 pure duodenal ulcers recorded by Moynihan, 4 had both hæmatemesis and mælena, 3 had hæmatemesis alone, and 4 had only mælena.

Loss of weight is common, and is probably often due to insufficient nourishment being taken, from fear of pain after ingestion of food.

The clinical types of duodenal ulcer may be divided into:

1. Those indistinguishable from gastric ulcer in symptoms and signs.
2. Those with hemorrhages from the stomach or bowel, or both, and with no other symptoms.
3. Those with symptoms resembling biliary colic, especially the pain, tenderness, and intermittent jaundice.
4. The perforative cases, which simulate also gall-bladder perforations and appendicitis.

Complications of Duodenal Ulcer.—These are numerous, and the most notable are: Perforation, periduodenitis, implication of the gall-bladder and bile-ducts or of the pancreas, cicatricial stenosis of the duodenum or the ampulla of Vater, with obstruction of the outflow of bile and pancreatic juice, compression of the portal vein, and cancer.

Perforation occurs frequently, probably in more than 50 per cent. (69 per cent., Collin; 43 per cent., Chvostek). Moynihan had 7 perforations in 52 cases operated upon (4 males, 3 females), and these were all in the first portion; Mayo had 6 out of 58. Of 131 collected cases with perforation, 91 per cent. were in the first part, 6 per cent. in the second part, and 3 per cent. in the third portion; 80 per cent. were in males. Nearly all the perforations are anterior (in 20 per cent. there are no preceding symptoms of ulcer). Weir had 9 perforation cases out of 34 without previous evidences of ulcer.

Diagnosis of Duodenal Ulcer.—Wrong diagnoses are frequently made, and most cases with perforation have been mistaken for appendicitis (19 out of 51, Moynihan). The mortality of perforation cases, while 80 per cent. in Brunner's time, is now much less, and within the past year 5 out of 7 of Moynihan's cases recovered, as did also 4 out of 6 of Mayo's.

The Course, Duration, and Prognosis of Gastric Ulcer.—Wirsung's statistics of 362 patients in the St. Hedwig's Krankenhaus showed that of those entering for treatment, 5 per cent. had been ill one week, 16.9 per cent. one to four weeks; 22.3 per cent., one to six months; 14.3 per cent., seven to twelve months; 19.2 per cent., one to two years; 10.9 per cent., three to five years; 9.4 per cent., five to ten years; and 1.9 per cent., ten to thirty years. The usual time limit was regarded as from three to five years, although Brinton records one case of thirty-five years' duration.

Lebert found 18 per cent. under one year; 46.5 per cent. from one to six years; 18 per cent. from six to twenty years; 2.5 per cent. from twenty to thirty-five years; 64 of Howard's cases averaged 4.1 years (two months to twenty years), and six were acute.

Recovery takes place in a majority of acute cases, provided perforation has not occurred, although definite statistics are unreliable because of faulty observations and diagnoses. MacCallum and Tull have drawn attention

to the long duration of the symptoms in their cases. In 18 of their series only two were acute. In 9 the duration had been from one to three years, in 4 the history lasted over a period of from five to ten years, and in 3 the duration was longer than ten years.

The mortality from all kinds of ulcers is variously given as 8 to 10 per cent. (Riegel), 10 per cent. (Lebert), 15 per cent. (Welch), and 25 per cent. (Leube); males were 22 per cent. and females 6 per cent. Debove and Raymond's cases showed 50 per cent. of deaths, 13 per cent. with perforation and peritonitis, 5 per cent. with fulminating hemorrhage, 20 per cent. with pulmonary tuberculosis, and 5 per cent. with inanition, while 7 per cent. had varying complications.

Russell, of England, has investigated the after-histories of cases of hemorrhage, and finds the direct mortality 2.1 per cent.; 4.3 per cent. died of intercurrent diseases; 42.6 per cent. recovered (27.7 per cent. had only one attack, and 14.9 per cent. recovered after one or more relapses); 6.4 per cent. were indefinite as to the prognosis and 44.7 per cent. still had gastric symptoms of varying intensity. Leube claims 50 to 75 per cent. of cures after four to five weeks of medical treatment, and holds that where cases are thereafter refractory, no medical treatment is of any avail, and surgical interference is justifiable. Lenhartz's results would seem to give even a more favorable outlook, and modern surgery, with its excellent cures through timely gastro-enterostomy, has entirely altered the former unfavorable statistics. Relapses are frequent, and may occur after several years. Einhorn's views are most encouraging as regards the prognosis of gastric ulcer. He draws attention to the fact that at postmortems one sees twice as many scars as fresh ulcers. Older ulcers are in some respects more favorable.

Sometimes a new ulcer forms alongside of an old and chronic one, and again sometimes cancer forms on the ulcer and quite alters the complexion of the case and the outlook.

How can we determine if an ulcer be healed or not? This is fraught with difficulties. In general, one may say that if the patient be free from digestive complaints, if pain be absent especially after solid food, if epigastric tenderness have disappeared, and if no occult blood be found after repeated tests in the fæces, the conclusion is reasonable that healing has occurred. This idea is strengthened by the additional evidence of normal weight and strength. Postmortem, however, one sometimes finds open ulcers which have given no symptoms for several years, showing that in spite of latency of all symptoms, healing may not be complete, or recurrence may have taken place.

Treatment of Gastric Ulcer.—Prophylaxis.—The etiology of gastric ulcer being still obscure, it is not easy to suggest along what lines the occurrence of ulcer may be prevented.

Locally the cells of the stomach are foci of lessened resistance, and it may be concluded that whether this be brought about by internal metabolic causes—by nerve influence or from injury to the mucous membrane in any way—the principle should be maintained of preserving the body in general from harm by maintaining good health and a normal nervous system, and protecting the stomach in particular by permitting only such foods as are unlikely to cause chemical or physical injury.

Early diagnosis is of paramount importance, not only to prevent complications later on, but to effect a cure. The acute and the chronic ulcers require different treatment, according to their physical conditions. For the simple

cases of so-called acute gastric ulcer, the strictest discipline is essential to a cure, and no neglect in hygiene, diet, or medicine is permissible. Failures in medical treatment come from indifference or incomplete discipline. Rest in bed is essential once the diagnosis has been established and the treatment commenced. By resting from at least four to six weeks in this way, hemorrhage and perforation may be prevented, and less of caloric units are required in the diet. After this the transition from rest in bed to walking about should be very gradual, and when once able to go about the patient should be made to rest after every meal. These precautions should be observed for some months or even longer; sometimes even this does not suffice, and yet one must insist that, apart from operative interference, rest to the body and to the stomach are the essentials of the medical treatment. Failures in medical treatment are no doubt very often due to lack of rigid control of rest and diet or to undue permission to relax from the severe regime which an ulcer cure should always entail.

Dieting.—Two rather opposed methods of dieting are recommended, the one with starvation, the other with feeding. There is no general rule of feeding applicable to all ulcer cases. Each case must be considered individually; some may require more, others less food; where the motor power is good and the stomach not readily contracted through spasms, a more generous diet may be recommended. If, on the other hand, the stomach is irritable and food causes pain and distress, it is well to treat such patients with as little food as possible and proceed very carefully in the transition to a more liberal diet.

In the so-called *abstinence cure*, during the first few days one depends exclusively upon rectal alimentation. This gives complete rest to the stomach, and may if necessary be kept up even for several weeks, provided some nutrition be maintained per rectum. Saline enemata alone may suffice, for as Edsall and Miller have shown, ordinary saline solutions given by the bowel will supply the very little that is necessary to replace tissue loss and keep up the caloric needs for some days or even weeks; indeed, starvation is good, and often well borne.

Saline injections are specially commendable because less gastric secretion is produced than when nutrient enemata are employed. One may, however, if preferred, give nutrient enemata, the patient receiving every six hours six ounces of broth with an egg and salt, and if desirable a little whisky may be added. These enemata should be given by means of the funnel and tube, slowly and by gravitation, and the bowels should be gently irrigated before every second or third enema.

Ewald recommends an enema made up of two or three eggs beaten up with one-half ounce of cold water, this mixture being added slowly to another made up of a few starch granules boiled in one-half cup of 20 per cent. grape sugar, to which two ounces of claret are added when lukewarm, the total being eight ounces. This should be introduced slowly high up, and should, if there be a previous cleansing saline injection, cause no irritation. This may be used twice or three times daily for from three to eight days. Water enemata may be used at intervals between the nutrient enemata if there be thirst, a dry tongue, or a bad breath.

Mouth Feeding.—The early administration of food by the mouth is apt to cause distention, increase the hyperacidity, induce vomiting, and thus directly or indirectly prevent the healing of the ulcer. It is a question

whether even a little water or chopped ice should be taken in the early days. According to some the practice is a bad one, because the water is not absorbed in the stomach, and therefore is apt to excite peristalsis and prevent the stomach from getting a complete rest. A little milk of magnesia or a mere mouth wash may suffice, and this will often help to prevent the not uncommon complication of parotitis which seems to be associated with dry mouths. Certainly one cannot prescribe general rules for all patients. When food is administered by the mouth it is well to commence with milk and lime-water, or albumin-water, or strained oatmeal gruel, at first in small quantities, a few ounces every two hours, and combine this as required with nutrient enemata. Whey is recommended by some. The milk may be given warm and diluted with one-eighth of lime water, or sodium bicarbonate; or it may be given, as suggested by Lambert, ice cold in teaspoonful doses. If curds form, one may add a little meal or flour to the milk, or peptonize it, or modify it according to Lambert's method. To many butter is tasteful in the early stages of treatment. Fütterer strongly recommends the early use of beef juice. After several days of this diet one may add broths and raw or soft-boiled eggs, and at the end of four weeks finely divided albuminous foods, such as minced meat, white fish, chicken, partridge, and tender beef, and a few light potatoes. One cannot lay too great stress upon the necessity of care in the *transition stages* from the liquid to the semisolid diet. After six weeks red meats may be given, and from that time on the greatest care should be taken for several months, even after an apparent cure has been obtained, and all coarse, irritating, and spicy rich foods should be avoided. Examination of the faeces from time to time for occult blood is an excellent guide for the transition to more generous diets.

Medicinal Treatment.—Perhaps the most universally employed drug in gastric ulcer is bismuth, which may be given as the subnitrate, or preferably the subgallate, in doses of from 20 to 30 grains (gm. 1.3 to 2) or more, several times daily. It is perhaps best suspended in gruel or barley-water. It may be given alone or combined with sodium bicarbonate or calcined magnesia, which both assuages the pain and burning and counteracts the hyperacidity. Bismuth, however, constipates, and may in this way be a source of trouble. The constipation may, however, be relieved by Carlsbad salts, given in doses of one dram in hot water in the early morning; or it may be better at first to give merely enemata of soapsuds or glycerin. Fleiner's bismuth cure consists of daily lavage and subsequent administration through the tube of six ounces of water with $2\frac{1}{2}$ to 5 drams of subnitrate of bismuth. The patient lies down for half an hour and then takes breakfast, this procedure being carried on for two weeks. It is very doubtful if the results and requirements justify a method of treatment so troublesome to the patient.

Silver nitrate is also highly recommended as an astringent and antacid. It may be given in pill form, $\frac{1}{4}$ to $\frac{1}{2}$ grain (gm. 0.016 to 0.03) per dose, or in solution with a few grains of sodium bicarbonate. Some prefer giving it through the stomach tube. Boas uses silver nitrate in solution of 4 grains to 4 ounces, and gives it in gradually increasing strength up to 7 grains to 4 ounces, adding peppermint to prevent nausea.

Lavage is carried out in cases where the stomach is intractable; and for ambulatory cases some sodium bicarbonate may be added to the water.

Treatment of Individual Symptoms.—For the *pain*, if severe, morphine may be necessary; a wet compress over the epigastrium is often soothing; orthoform may be used with some benefit. For the *vomiting*, ice, bismuth, nitrate of silver, cocaine, and opium are recommended. For the *anæmia*, albuminate of iron may be given, as Ewald suggests, by adding 1 dram (4 cc.) of a 2 per cent. solution of iron sesquichloride to 2 ounces of albumin water; or Blaud's pills with a laxative may be used. For the *hamatemesis*, absolute rest and quiet, ice to suck, and small doses of adrenalin chloride, 10 to 20 drops, three or four times a day, of the 1 to 1000 solution, may be effective. Lucas Championnière seems to have shown that lavage is good because the stomach is not in repose during hemorrhage, and washing out gives it the necessary rest. Ewald used ice water for the lavage, and others again recommend gelatin water. It is thought by some that high enemata of hot water, acting reflexly, may check the hemorrhage and combat the shock. It is well, perhaps, both for the restlessness and for the quieting of the circulation, to use morphine hypodermically. For the severe recurrent hemorrhages surgical intervention may be necessary and is often most successful.

Other Methods.—As has already been stated no single rule applies to all cases. Each patient according to the constitution, the symptoms, history, and progress of the disease will require treatment appropriate to the condition.

Leube's Method.—The patient is placed in bed for ten days, the epigastrium washed with alcohol and sublimate solution, then boracic ointment is applied on a cloth, and over this a hot flaxseed poultice, 20 x 10 cm., every fifteen minutes, for ten hours during the day; at night a wet compress is applied. This treatment is carried on for ten days. Then a simple cold compress is applied at night during the next three weeks, while in the daytime an abdominal flannel binder is worn. During convalescence the patient rests for two hours after meals, not even doing sewing, etc. The only contra-indications for poultices are menstruation and recent hemorrhage (within three months). When recent hemorrhage has occurred, or when the stools show traces of blood, an ice-bag may be applied instead of the cold compresses. One pint of Carlsbad water is drunk slowly in the morning for one month and alkaline waters are taken during the day. Few drugs are used, unless bismuth or sodium bicarbonate. The constipation is treated by enemata of tepid water, by Carlsbad salts, or, after the eleventh day, 1 dram (gm. 4) of a powder consisting of pulv. rhei., 20 parts; sodii sulphat., 15 parts; sodii bicarb., 7½ parts, is given. The diet during this treatment is as follows: For the first ten days he recommends boiled milk and Leube's meat solution and soft, unsweetened zwieback (rusk). Then for a week, rice or sago soups, boiled with milk or white of egg; soft-boiled or raw eggs. Later, other tender meats, and after the fifth week careful ordinary dieting. Leube claims in this way to have cured 74.1 per cent. and improved 21.9 per cent; 1.6 per cent. were not relieved, and 2.4 per cent. died. F. P. Henry has recently confirmed the excellence of this method.

Lenhartz's Method.—Basing his views on the theory that ulcer is prevented from healing because of mechanical stretching of the organ, hyperacidity, and anæmia, Lenhartz has opposed the main ideas of the usual "abstinence cure." Milk alone he regards as insufficient, all the more so as the anæmia is thereby aggravated, and if the bulk of milk be increased to a general caloric sufficiency, stretching of the stomach results. Lenhartz, therefore, advo-

cates: (1) Continuous administration of concentrated albuminous food which will use up the hydrochloric acid physiologically and prevent its unfavorably influencing the healing of the ulcer. (2) Rapid increase of nourishment to repair lost strength and thus indirectly aid the healing process. (3) Absolute rest in bed for four weeks and prevention of distention by means of an ice-bag for the first ten days. Bismuth subnitrate, 30 grains (gm. 2) daily, and careful limitation of fluids. Morphine is unnecessary. Sometimes silver nitrate is useful, and iron later on. Occasional glycerin enemata may be given after the first week.

To carry out this idea, fresh eggs are the main article of diet, given in increasing quantities each day, chiefly raw and beaten up. One to three eggs are given on the first day, and one added on each subsequent day, until eight are taken daily, and this number maintained. They may be given iced in teaspoonfuls, and later soft boiled. Sugar is added to the eggs on the third or fourth day, in gradually increasing amount (30 to 50 gm.). Iced milk must be given in extremely small amount, only increased with the greatest care to obviate unnecessary distention, which Lenhartz regards as the most serious of all ordinary events which may obviate the healing process (200 cc. are given at first and 100 cc. added daily up to 1 liter). Eggs alone being insufficient, finely divided meat, preferably raw, or scraped beef, is given from the sixth day onward (at first 35 to 70 gm.). On the seventh day, soft-boiled rice is given, and on the eighth day, toast, softened rusks, or cereals, and 20 to 40 gm. of butter, and finely divided raw ham on the tenth. Strong broths are harmful on account of their extractives and spices, which tend to increase hyperacidity.

Among the benefits of the cure, Lenhartz specially cites: (1) The short duration of treatment. (2) By means of dieting alone the pain was relieved, and opiates and narcotics were never required after the first four or five days. (3) Vomiting likewise ceased. (4) The number of recurrent hemorrhages was far less than by other modes of treatment. Among 201 cases only 13, *i.e.*, 6.4 per cent., had recurrent hemorrhages; and the later cases treated after greater experience of the method showed increasing benefit. Thus out of the last 106 cases, only 4 showed recurrence of the bleeding. (5) Quick return of strength and working capacity. (6) The good effects are lasting.

The treatment is begun at once, in order to bind the hydrochloric acid and prevent its evil effects on the recent thrombus. Absolute rest is maintained by this means better than by the abstinence cure, for even with an empty stomach some peristalsis takes place and is only likely to be increased by the effects of unused hydrochloric acid, especially if in excess.

In advocating his method, Lenhartz and others of his views have stated that by use of the abstinence cure patients suffer greatly through loss of weight and strength, that the blood pressure and volume of the pulse sink rapidly; while some opponents find from personal observations that the contrary is the case, *viz.*, that the heart's power remains good, that the pulse is never dicrotic, that the blood pressure never sinks more than 30 mm., and that the urine remains fair in quantity. Neither are the red cells found to alter greatly through the abstinence cure.

Senator adopts a middle course and argues that one should not distress the stomach by an excessive amount or weight of food, that a soothing diet is useful to prevent recurrence of the hemorrhage, that a diet which will

counteract the hyperacidity is useful, and that the food administered should be easily digested and nutritious. To this end he believes that gluten, fat, and sugar fulfil all these requirements. For this purpose he recommends lerin, or its modification gluten, or one may use gelatin because of its hæmostatic effect. Fat is useful because of its counteraction on the acids and its soothing effect on the surface of the ulcer.

He allows fresh butter and cream in small amounts often, *e. g.*, 30 gm. of butter and 250 cc. of cream in twenty-four hours. If the butter is not well borne, it may be given iced in lumps to suck or swallow. Cream is given iced and beaten up. These quantities may be gradually increased if no hemorrhage occurs, and one may add milk, beaten up eggs, and meat, as recommended by Lenhartz. Gelatin should be stopped after a while because of its constipating effect, and in its place one may use jellies. Instead of butter, good oil may be given, either iced or as an emulsion (almond emulsion or almond milk).

Rosenheim likewise approves of gelatin, using it in 5 to 8 per cent. solution in citric acid, and avoids meat, preferring a lactovegetable diet.

Cohnheim's oil method has much to recommend it. Two to four ounces (60 to 120 cc.) of olive oil are given by means of the tube daily before breakfast, or one to two ounces (30 to 60 cc.) without the tube before each meal. This soothes, protects, and enables the patient to take more food and exercise. *Kraus* adds 75 grains (gm. 5) of sodium bicarbonate to 3 ounces (90 cc.) of the oil, and gives it in $\frac{1}{2}$ ounce (15 cc.) doses before meals.

Chronic ulcers which have failed to respond satisfactorily to medical treatment are in some cases benefited by surgical interference; others, again, do not admit of operation because of associated gastric and other abdominal conditions which render the operation unsuccessful and merely aggravate the already existing misery.

Surgical Considerations.—Many statistics have been compiled with a view to demonstrate the superiority of medical over surgical methods of treatment, and vice versa. Such proofs, however, scarcely carry much weight when diagnoses are often uncertain, when conditions and symptoms vary within the widest of limits, and when the individual circumstances make it always imperative to consider each case by itself and for itself, rather than to place it under any very general category. Many cases certainly fail under medical treatment, and just what particular circumstances interfere with the healing of so many simple ulcers, even when seen in early stages, is as yet unknown. Certainty statistics now in use are for the most part unreliable and more or less useless.

Sears, of Boston, synopsised the results in 183 cases medically treated: 42 had been discharged as well, and of these, in 15 recurrence was known to have taken place, 105 were relieved, 9 were unrelieved, and 27 died (but only 13 as the result of ulcer).

Friedenwald reported on 287 cases variously treated as follows:

	Rest cure	Ambulatory	Operation
Cured	67	123	6
Not cured	8	75	
Died	3	0	4

One must distinguish between a medical treatment in which recovery has occurred after a few weeks and one in which no recurrence is known to

have taken place after several years. In the former instance one cannot speak of a cure, for the condition may be simply latent for months and simulate a cure. Hence, again, the wisdom of not being too much influenced by statistics of this kind. Paterson and Rhodes, for example, estimate medical cures as being less than 25 per cent., and Bulstrode found that of 500 cases in the London Hospital, 82 per cent. had been discharged as improved or cured, and that 40 per cent. of these had relapsed. Russell, of Birmingham, in studying the after-histories of 47 ulcer cases, found that 42.6 per cent. had been cured, and that 44.7 per cent. were still suffering. Schultze, who reported at one time on 291 cases, of which 89 per cent. had been discharged as well or improved, gave the subsequent history in 157 of these patients. Only 53 per cent. were well, 23.5 per cent. were still suffering somewhat, and 15.4 per cent. were very poorly; 7.6 per cent. had died.

Medical failures, then, are variously estimated as: 11 per cent., Friedenwald; 21 per cent., Sears; 44.7 per cent., Russell; 47 per cent., Schultze; 50 to 60 per cent., Bulstrode; 50 per cent., Debove; 74 per cent., Leube; 75 per cent., Ewald; 75 per cent., Greenough and Joslin; and 85 per cent., Welch. The actual mortality under medical care is evidently subject to wide variations, and reliable statistics give in some series 1 per cent.; in others as high as 22 per cent. (Stoll) and 20.5 per cent. (Gilman Thompson). The average mortality under medical treatment in 20 large series of statistics collected and quoted by Musser is about 8 per cent.

Surgical interference, on the other hand, shows an ever diminishing mortality in each succeeding year since operations on the stomach have become common. While in earlier years operations were frequently repeated because of incomplete results, the number of these likewise attains an ever diminishing proportion in accord with the more judicious selection of cases and better methods of operation. Repetition of operation was necessitated either from recurrence of old ulcers, from formation of new ones near the site of operation, persistence of old symptoms, the development of vicious circles, or hernia, but the proportion of these has rapidly lessened.

The following figures present the case as it now stands with modern surgery: Hartmann, 1895 to 1899, 23 cases, 6 deaths, 25 per cent. Heydenreich estimated the surgical mortality at 16.2 per cent. Moynihan's yearly statistics are most instructive. In 1904, among 100 gastro-enterostomies for simple chronic gastric or duodenal ulcer (including cases of hemorrhage), there were 2 deaths (2 per cent.). Up to April, 1905, among 153 gastro-enterostomies, etc. (two only requiring second operation), there were 2 deaths (1.5 per cent.). Later, among 206 gastro-enterostomies, etc., including hemorrhagic cases and hour-glass stomach, there were 9 deaths (4.3 per cent.). In May, 1906, among 251 gastro-enterostomies, etc., there were 9 deaths (3.5 per cent.). Mayo Robson, among 210 gastro-enterostomies, etc., had 8 deaths (3.8 per cent.).

Comparing these figures with those of earlier years, the improvement is striking, and comparing them with the mortality by medical treatment alone, the benefit attributable to surgery is enormous.

Kreuzer, in Zürich, recently recorded his observations on the after-effects of operations for gastric ulcer, and arrived at the following conclusions: (1) Where before operation dilatation had existed, this had nearly or quite disappeared soon after gastro-enterostomy had been performed.

(2) Secretions which were disturbed before were later normal. (3) Hyperacidity gave way to normal acidity or subacidity. (4) Reflux of bile and pancreatic juice into the stomach gave rise to no serious complaints or results.

Surgical Methods and Indications.—Various methods are adopted in the operations for simple chronic gastric ulcer, that most favored by surgeons being simple posterior gastro-enterostomy. This was first done for cancerous stenosis of the pylorus by Wölfler, on suggestion of Nicoladoni, in 1881, and first for ulcer by Doyen in 1892. Mikulicz believed that where excision was easily done this was the proper procedure, not only to avoid recurrence of ulcer in this area, but to preclude subsequent malignancy. With this, a posterior gastro-enterostomy is advisable on account of the possible presence of multiple ulcers, the later formation of strictures at the excised area, and the better drainage of the stomach, which would aid in the healing of the gastric wound and reduce pyloric spasm.

Mayo's results with gastric jejunostomy are especially encouraging. Pyloroplasty and gastroduodenostomy have found less favor in the eyes of most surgeons, the former, because of the necessity for operating through infected and indurated tissue, the latter because, if used in open ulcer cases, the food must pass over the ulcerated area to gain an exit.

The indications for surgical interference may be briefly synopsisized as follows: (a) Chronic gastric ulcer which resists medical care, as implied in the proper rest, diet, and medicinal agents, and which after two months is still accompanied by persistent nausea, vomiting, and pain, or which through varying conditions induces prolonged suffering and misery, with patients failing in health and more or less incapacitated. Here operation is indicated, and all the more so in view of the unknown and unforeseen but common complications, of which the more serious are perforation, hemorrhage, perigastritis, with adhesions or abscess, stenosis of an orifice, and constrictions causing deformities.

(b) Gastric ulcer which, after causing mild dyspeptic signs over a prolonged period, culminates in hæmatemesis.

(c) Chronic recurring ulcers, even when each attack lasts over a comparatively short period.

The contra-indications for surgical treatment are: (a) Acute non-perforating ulcer (called also non-indurated ulcer); (b) neurotic individuals with marked gastropotosis; (c) simple dilatation without stenosis, especially if with enteroptosis.

Much has been written within the last decade as to the pros and cons of surgical intervention in these cases, and there is overwhelming evidence to prove the benefits accruing from modern surgical methods, which cure many patients not amenable to medical treatment.

The rule laid down by Dieulafoy, that operation was necessary if the loss of blood exceeded one-half liter, is not sustained, but when possible it is well to operate in the intervals between the hemorrhages. The operations recommended are of two kinds:

1. Direct, in which the bleeding point is determined and either ligatured or cauterized; or, better still, the ulcer excised. Ligature is not always possible; one may not always find the bleeding point, or the search for it may prolong the operation and endanger the patient's life still more, or several ulcers may be present and not recognized, and thus only one possible

source of the hemorrhage be controlled. For this reason it is recommended that unless the bleeding point is readily reached and fixed, no delay should be permitted in the effort to find it, although if the ulcer be easily found and excised the additional safety thereby precludes a recurrence of hemorrhage from at least one source, and one may then proceed in addition to the indirect method. This depends, however, on the site of the ulcer and when the necessity arises for insuring satisfactory drainage.

2. Indirect, *i. e.*, by gastro-enterostomy. This has numerous advantages. In most cases the hemorrhage ceases, probably by relieving the distention in the stomach (which of itself may keep the ulcer disturbed and tense and thus maintain patency of the vessel).

It is a rapid method and easily done, and thus safe, causing but little shock. Then, too, on account of the short circuit the organ is emptied quickly and completely, and thus allowed to contract. Healing is thus aided, pyloric spasm is lessened, more food is taken in, and perhaps even the necrotic process itself diminishes.

Surgical Treatment of Perforation.—Howard states that 8 cases have been recorded as recovering after generalized peritonitis without operation, and Carless' statistics, which seem optimistic, give 4 per cent. recovering under similar conditions. The early surgical treatment as a result of early diagnosis and perfected technique has given brilliant results, and the following statistics afford ample evidence of the success of more modern methods: Mikulicz, 1885 to 1893, had 35 cases with 34 deaths, *i. e.*, 1 recovery, and from 1894 to 1896, 68 cases with 36 deaths, *i. e.*, 32 recoveries. Tinker in 1900 recorded 232 cases, with 113 recoveries, and Robson in 1900 recorded 429 cases, with 279 recoveries (65 per cent.). J. Bell, of Montreal, in 1903, had 10 cases, with no deaths.

The proportion of recoveries has a direct relation to the early time of operation after rupture; the sooner operation is done, the greater proportion of recoveries.

The multiplicity of ulcers is of the greatest importance, and is to be considered in all operations for perforation, and in not a few cases a second operation has been necessary for this reason, when the first had healed. Hence the recommendation of some surgeons to perform a gastro-enterostomy in all cases after closing the perforation, in this way obviating possible extension of other ulcers.

Surgical Treatment of Hæmatemesis.—While general indications may be given for operation in cases of gastric ulcer with hemorrhage, each patient must be regarded individually, and due consideration must be taken of the past history, present state, opportunities for good surgery, etc. In a general way it may be said that two classes of cases call for operation: (1) Hæmatemesis following upon a prolonged dyspepsia. In these cases the ulcer is in all probability an old one, and the tissues in which the vessel lies indurated. Here spontaneous healing is more difficult, the vessel is less elastic, and thrombosis occurs less readily. (2) Persistent recurring hemorrhages, whether small or large, especially when the intervals are not long enough to permit the patient to regain lost ground. Here, too, the ulcer is chronic.

Acute cases, especially in comparatively young individuals, in whom one or two large bleedings occur, do not usually require surgical treatment, and the condition improves by mere rest and absence of treatment, assisted at

times by such medical treatment as withholding of food and the use of morphine and styptics. It has been estimated that 93 to 97 per cent. of patients with these large acute hemorrhages get well if left alone, so that operative intervention is to be regarded as adding a complication rather than effecting a cure.

Hour-glass Contraction.—The treatment of hour-glass contraction is essentially operative, the object being first to drain both sacs and allow of healing, which, however, involves a double operation. Many methods are recommended:

1. Gastroplasty, which affords a better communication between the two sacs, is a suitable operation for the simpler cases with little infiltration. It is done with or without gastro-enterostomy, according to the requirements.

2. Gastro-gastrostomy, in which the two sacs are brought together and directly joined, is often commendable in patients whose hour-glass contraction is extreme, and where the opening between the sacs is very small.

3. Partial gastrectomy, which removes the total area of disease and unites the divided healthy ends of the stomach, is the ideal operation, because it corrects the deformity as well as eliminates all the old or any recent disease.

4. Gastro-enterostomy alone is of little use. To be beneficial it should be double, and this makes a serious operation and affords some danger of a double vicious circle.

Mayo Robson reports 19 cases of simple hour-glass stomach, in 11 of which he did gastroplasty, with recoveries in all. In one, a fatal case, he performed gastroplasty and pyloroplasty combined; in another, also fatal, an anterior gastro-enterostomy. In the 6 others a posterior gastro-enterostomy was performed, followed by complete recovery; his total being 17 recoveries out of 19 operations. Moynihan's results are equally satisfactory: out of 20 cases he had but 3 deaths. In 7 a gastroplasty was done, in 6 gastro-enterostomy, in 2 a combination of these two methods, in 1 a gastro-gastrostomy, in 3 a gastro-enterostomy combined with gastro-gastrostomy, and in 1 a dilatation of the stenosis.

The necessity for anastomosis is largely determined by the situation of the ulcer, its extent, and its liability to induce stenosis, adhesions, and deformities.

Moynihan's statistics show that out of his 24 patients, 7 had required a gastro-enterostomy at the time of operation or at some later date. The new opening affords better drainage, and thus aids in the healing of the ulcer. The distention is also relieved, and this, again, renders the patient more secure against hemorrhage. Lastly, the operation permits of the earlier administration of food.

CANCER OF THE STOMACH.

Definition.—A malignant epithelial neoplasm of the stomach.

Incidence.—About one-half of all cancers arise in the stomach. Combined statistics of 1,000,000 hospital *admissions* show 4700 gastric cancers, *i. e.*, 0.47 per cent. In combined *autopsy* statistics of over 50,000 cases there were over 2000 gastric cancers, *i. e.*, 4 per cent. Reiche's Hamburg statistics (1872 to 1895) show that 50.2 per cent. of all cancers are gastric, and that cancer of the intestinal tract, as a whole, forms 75 to 85 per cent. of all cancers. Combined statistics of 70,000 cancer cases collected from various series

show over 21,000 as gastric, *i. e.*, 33 per cent. Cancer of the stomach is increasing in frequency, or is being more often recognized by better methods of diagnosis.

Regional.—Deaths per 1000 from cancer in general vary within rather wide limits: Amsterdam, 12; Berlin, 22.4; Frankfort, 47.6; Geneva, 53; St. Petersburg, 15; Christiania, 29; Copenhagen, 33.2; New York, 19.3; London, 28.7; and Edinburgh, 25.4.

Climatic conditions seem to have but little influence except that in tropical countries gastric cancer is more uncommon, *e. g.*, in Egypt (Griesinger). Regional differences where they exist are probably associated more with the manner of living and the diet than with the climate.

Race.—Gastric cancer is much less common in negroes; the proportion is one-third less in New York among 7518 negro deaths. Osler and McCrae found the proportion of whites to negroes as 131 to 19, and the proportion of admissions was 6 to 1. In 1880 the death rate in the United States from all cancers was: whites, 25.9; negroes, 12.6.

Etiology.—**Age.**—The following results appear from 7000 cases gathered from combined statistics:

Ages.	Per cent.
10 to 20	0.08
20 to 30	1.5
30 to 40	8.8
40 to 50	18.0
50 to 60	28.0
60 to 70	28.0
70 to 80	14.0
Over 80	2.0+

Congenital cases have been described (Williamson and Widerhofer), and Cullingworth performed an autopsy on a child, aged five weeks, in which gastric cancer was found. Osler and McCrae found 10 cases in the literature under ten years of age, and 13 cases between ten and twenty years. Probably more than 2 per cent. of all gastric cancers occur under thirty years of age. Hahn found 50 per cent. of cases over sixty years of age—so also did others; at all events, probably 80 per cent. or more occur between the ages of forty and seventy. With better methods of examination, however, more and more cases are found among youths, which alters the ratio somewhat and brings the average down to earlier years. At the Royal Victoria Hospital Montreal, over 50 per cent. were between the ages of forty-five and fifty-five years.

Sex.—Of 20,000 cases collected from my combined statistics, 58 per cent. were in men. Welch gives the relation as five males to four females.

The relation of sex and age may be gathered from the table of Perry and Shaw according to decades:

Decades.	Males.	Females.
Second	2	1
Third	3	4
Fourth	18	16
Fifth	76	26
Sixth	60	23
Seventh	39	5
Eighth	6	3
Ninth	0	1
Total	204	79

At the Royal Victoria Hospital the average age among females was 59.71, and among the males 53.7.

Heredity.—Opinions vary widely and statistics are very incomplete. In 1075 cases Snow found heredity in 167, *i. e.*, 15.5 per cent.; Schule gives 6.5 per cent. as hereditary, Lebert 7 per cent., and Roth 50 per cent.

Of 2700 cases of gastric cancers, 12 per cent. showed an hereditary history. In the Royal Victoria Hospital, 24 per cent. gave a probable hereditary history, which was undoubted in 18 per cent. Cancer as a family affection perhaps occurs in 10 per cent. in all cases. At the same time, gastric cancer being in any case a frequent disease, there can be little that is reliable in such findings. Just how frequently the offspring of cancerous patients have subsequently developed the disease we do not know, and our histories refer to relationships which are often too remote and indefinite to permit of accurate conclusions.

Previous Gastric Disease.—Long-continued irritation as a cause of cancer of the stomach is extremely dubious, and badly used stomachs seem to suffer no more than do those well treated. There is no definite information which leads us to suppose that gastritis leads to carcinoma; in fact, as Riegel suggests, in a large proportion of cases the development of cancer is almost sudden in the midst of complete health. Yet where previous dyspepsia has existed, it is not easy to determine how far this was due to an already existing cancer and how far a chronic gastritis or chronic ulcer was responsible for the symptoms. Of course, where the dyspeptic symptoms have existed for years, it would be easy enough to exclude cancer. Thus, for example, out of 83 cases at the Royal Victoria Hospital, 15 had chronic dyspepsia for years previously, and of these there were 6 that had had signs suggestive of previous ulcer.

Chronic Ulcer and Cancer.—Wide variations in the statistics are noted, consequent on their dependence upon many views and imperfect observations as to the presence of ulcer. Cruveilhier discussed the relation of cancer and ulcer in 1839, and Rokitansky soon after recognized its possible implantation in ulcer. Dittrich, in 1848, recorded 160 cases of cancer, of which 6 were of this etiology. Haberlin gives 10 out of 138 cancers developing on the site of an ulcer, Dittrich 6 out of 160, Rosenheim 4 out of 56, Montreal General Hospital (1880) 1 out of 28, and Perry and Shaw give 9 out of 100, giving a total of 30 among 482, or 6.23 per cent.

This percentage coincides with other earlier averages (Barling and Hirsch, 5 per cent.; White, 5 to 9 per cent.; Lebert, 9 per cent.), but later observations differ markedly, and Robson estimates the number at 59.3 per cent., while Zenker thinks that nearly all cancers develop from ulcers. Riegel observes that by far the greater portion of the stomach (*i. e.*, fundus, anterior wall, greater curve, cardia) is affected by ulcer (only 20 per cent.), and similarly in cancer these parts are less commonly involved at the onset. Robson and Moynihan likewise find these regions cancerous in only 16 per cent., Graham, working in the Mayos' clinic, found 81 out of 135 patients with cancer had a history of ulcer. Ewald, on the other hand, observes cancer of the orifices in 70 to 75 per cent., while ulcer affects the orifices in 16 to 18 per cent. only.

Fütterer concluded that cancer frequently formed at the edge of ulcers especially where the part was most exposed to irritation, that in the pyloric region this development occurs in the lower margin. But if the position

of the organ alters from dilatation or adhesions, other parts of the edges may be involved. Cancer develops on ulcer most frequently in the pylorus.

Alcohol.—Fifty-six per cent. of the Royal Victoria Hospital cases were habitués, and of these, 20 per cent. were hard drinkers, and 30 per cent. drank moderately but constantly.

Trauma.—In 62 cases of cancer of the intestinal tract, Boas found 9 with a clear history of antecedent trauma, and *only 1 in gastric carcinoma*. According to some, and especially Fürbringer, trauma has a definite effect in aiding the development of latent cancers and increasing the rapidity of their progress. Kuttner and Lindner, after careful inquiry, found no history of injury in 66 consecutive cases.

Brosch endeavored to show that the frequency of cancer at the pylorus and on the lesser curvature was due to its slight degree of mobility and its lesser tendency to yield to blows upon that region, in contrast to the mobile greater curvature. The resulting scars he regarded as predisposing causes of cancer. The frequency, however, with which gastric ulcer occurs on these portions of the stomach would probably afford the simpler reason for the development of cancer in that region, besides which is the fact that these parts are more exposed to injury and irritation from within the stomach. Erosions from whatever cause are also regarded by some as causes, and doubtless have a similar relation to cancer as do chronic ulcers.

Auto-inoculation.—Transplantation of cancer from a primary growth on the tongue into the stomach has been recorded by Klebs, Cornil, and others, but such instances must be very rare.

The possibility of auto-inoculation from one cancerous tissue to another has been frequently noted. This has been the subject of observations among surgeons who note the line of incision or of puncture in abdominal cancers infected from the original neoplasm. Cripps, Fenwick, Schimmelbusch, and many others record the possibilities of conveying cancer from one part of the body to another, as has been already demonstrated in guinea-pigs.

Social Position.—Perhaps where poverty exists and consequently excess of vegetable diet is indulged in, the stomach thus persistently injured and irritated may be predisposed to cancer. Eichhorst, at all events, favored such a view.

Pathology and Morbid Anatomy.—Cancer of the stomach is a malignant epithelial overgrowth extending from the secreting cells of the mucosa to the other coats of the organ, and thence usually to neighboring tissues, glands, and organs, or to distant parts by continuity, contiguity, and metastases through lymphatic and blood channels. It is nearly always primary in this organ.

Secondary cancer of the stomach is rare. Combined statistics show the proportion to be about 1.1 per cent., *i. e.*, 5 cases out of 440, although Hale White regards the proportion as 6 to 7 per cent.

It arises either by contiguity or by metastases from distant viscera. Involvement by contiguity from the pancreas, liver, œsophagus, etc., is, of course, not infrequently noted, and even transplantation from the tongue has been at times recorded, with the development of a secondary epithelioma of the stomach as a direct result of swallowing detached particles of the tumor.

Site of Growth.—While the pylorus has usually been regarded as the most common place of origin, it is now conceded by many that the lesser

curvature shares this peculiarity with it. Boas found in 125 cases, 34 in the pylorus (27.2 per cent.) and 33 cases (26.4 per cent.) in the lesser curvature; Tabora found 39 per cent. in the lesser curvature. This fact is of importance in regard to diagnosis, for it gives a reason for the lesser frequency of classical signs and symptoms of obstruction, and it emphasizes the need of examining for other evidences of cancer of the stomach than merely those of obstruction in order to obtain earlier diagnosis. Statistics of past years are comparatively useless and unsatisfactory because taken from all cases, not only the early ones but also from those advanced, and at autopsies the exact origin is in most instances uncertain. It is therefore of little value to know that of 5444 gastric cancers, 3319 (nearly 61 per cent.) were said to have been pyloric. It is *more to the surgeon than to the pathologist* that we must look for a knowledge of the facts.

Newer observations tend to show that the former theory that most cancers begin at the orifices of the stomach is incorrect, and that these are more often secondarily involved. Such was the conclusion of Israel and also of Hanseemann. Mikulicz and Kausch in 1900 mention the probability that 40 per cent. of cases of cancer of the stomach begin in the lesser curvature, their observations depending on laparotomies and thus representing earlier types than are seen by the pathologist.

Boas thinks that a common site of origin is the *vicinity* of the pylorus, *i. e.*, to the right of the scrobiculus cordis or else in it. In 40 cancers recently recorded he found 25 on the lesser curvature and only 6 at the pylorus. These observations have given rise to the statement of Brosch that a good explanation exists in the fact that the lesser curvature, lying as it does in the line of passage of foodstuffs, is exposed to irritation and injury, and is therefore more vulnerable to the development of cancer.

As regards other sites of origin, two series may be quoted:

	Pylorus and lesser curvature. Per cent.	Cardia. Per cent.	Diffuse. Per cent.	Anterior or posterior wall. Per cent.	Greater curvature. Per cent.
Lebert and Hahn	51	29	15	0.08	0.04
Orth	80	10	10		

Multiple Cancers.—Occasionally several tumors are found in the same stomach, *e. g.*, Maurizio saw one case with cancer at the pylorus and at the same time one at the cardiac end, and Gierke reported a case with four cancers in the one stomach.

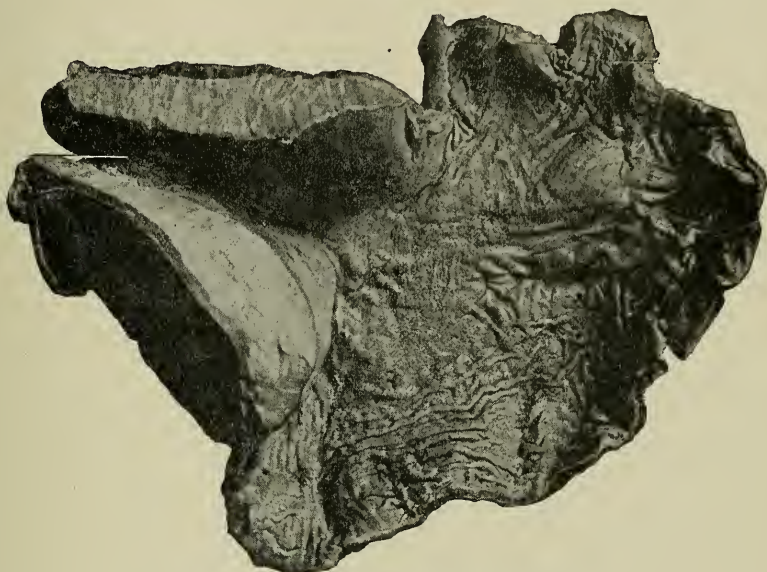
Relation of Cancer to the Changes in the Mucosa.—The general mucosa elsewhere suffers coincidentally with the progress of the cancer. Usually the superficial cells are fairly preserved or more goblet cells appear. The peptic glands become mucous glands, the border cells disappear more than the chief ones, and all are more or less surrounded by new small round cells. The mucous membranes may be completely absent in some places, due to the development of a general atrophic gastritis with small-celled infiltration, cysts, etc.

There is little to substantiate the view of Hayem and Mathieu that cancer develops secondarily on atrophic gastritis. Usually where the glands are preserved, the secretion of hydrochloric acid persists, and where, on the other hand, there is diminution of acid secretion, there is also extensive change in the secreting cells. It is interesting to note that secretion which has dis-

appeared may in rare cases return after resection of the cancer, but not after mere gastro-enterostomy, showing that while the cancer exists and develops, the regeneration of secretion is less likely to occur.

The observation of Fenwick is noteworthy, viz., that with cancers of organs other than the stomach there may be absence of normal acid gastric juice. Friedenwald and Rosenthal found a similar result in 9 out of 29 patients. Their figures were briefly as follows: the cases included 9 breast cancers, 5 in the uterus, 7 in the rectum, 2 of the tongue, 4 of the face, 1 of the intestines, and 1 of the pancreas. In 9 there was low total acidity and no free HCl. In 10 there was low total acidity and diminished HCl, and in the remaining 10 the secretion was normal.

FIG. 4



Scirrhus carcinoma of pyloric end of stomach.

The Cancer may be Circumscribed or Diffuse.—When circumscribed, the tumor usually projects as a fungating mass with a villous surface, or one which is more smooth and flat, and showing central dimples or areas of degeneration and ulceration. The degeneration has no necessary relation to the size of the growth, and may occur early in small neoplasms, or only much later after the mass has attained very extensive proportions.

The diffuse variety extends mainly in the submucosa, which thus becomes thickened, giving to the whole wall a greater rigidity and firmness and making it much more resistant to the knife. The mucous membrane itself is usually roughened and in parts degenerated, while the organ is either large and dilated (if pyloric stenosis has occurred), or shrunken and the cavity much smaller than normal. In some of these diffuse cancers it may be impossible to distinguish the condition from a mere cirrhosis (interstitial gastritis) except by microscopic examination, and often even then it is not

easy to find the few areas of cancerous growth amid the abundant dense fibrous tissue.

Two main types occur: The *scirrhus* and the *medullary*, although transitions are found in a large proportion of cases. Each of these may undergo more or less extensive *colloid* change, giving a third variety.

The *scirrhus* cancers are perhaps the most common, although transition forms and mixed types are usually present in nearly all gastric cancers. They are, as a rule, hard tumors, due to the preponderance of fibrous tissue (where epithelium and stroma preserve the alveolar arrangement the name *carcinoma simplex* is used). The wall is thickened, the extension is often diffuse rather than circumscribed, and there is a tendency to scar formation in some places, while in others superficial ulceration is seen. Scirrhus cancers are slow growing, spread gradually into the surrounding tissues, especially the muscle. It is in this form that differentiation is sometimes difficult between cancer and simple chronic indurated ulcer, or, again, cirrhosis ventriculi. Scirrhus neoplasms are commonest at the pylorus, so that stenosis occurs, owing to the increasing size of the tumor or to shrinkage and rigidity at the orifice.

The *medullary form* is soft, very cellular, and thus on section shows more "juice." The progress is more rapid, degeneration and hemorrhage more common, and metastases are more readily formed. The glands especially are subject to invasion.

Papillary outgrowths of a special *adenocarcinomatous form* are not uncommon. They form soft, fungating excrescences, oftenest at the pylorus, where they are sharply delimited at the duodenal end. Ulceration, necrosis, hemorrhage, and even perforation are apt to occur with this special variety.

The *colloid changes* may supervene in any form of gastric cancer. Diffuse thickening occurs from the presence of yellowish-brown, jelly-like, translucent fluid, which is seen occupying large or small spaces in the neoplasm. It is essentially a degenerative process of the cells and tends to invade the omentum, but not, as a rule, the intestines.

Myxomatous degeneration may occur in the fibrous stroma of any cancerous tumor.

Squamous-celled cancer commonly occurs about the cardia as the result of an epithelioma commencing in the œsophagus. It is rarely elsewhere in the stomach, except when secondary nodules form there either by transplantation from squamous-celled tissues (tongue, œsophagus) or by metastases from the œsophagus, as has been described by Klotz and others.

Extension and Dissemination.—The disease may be *confined to the stomach*, and may induce ulceration, degenerative changes, hemorrhages, etc. Extension in the stomach occurs chiefly by the lymphatics running in the fibrous intersections between the muscle bundles. Here the course is vertical toward the serosa, which may also be involved. As Cunéo has shown, the gastric lymphatics run toward the glands along the lesser and greater curvatures, more especially toward the pyloric end and in the adjoining portions of the great omentum, and thence through the cœliac glands on their way to the thoracic duct, thence to the supraclavicular region, as well as to the subclavian vein and the general circulation. In pyloric cancer the lesser curvature is rapidly invaded, and the lymphatics and glands become involved as far as the point where the coronary artery joins the stomach; here the lymphatics pass *from* the lesser curvature. The lymph nodes of the

greater curvature do not usually go farther to the left than a point near the median line. The fundus is almost devoid of lymphatics.

The disease commonly spreads in the subserosa and if degeneration occurs here, ulceration and even *perforation* may follow with a consecutive peritonitis, local with adhesions, or more general. Owing to the slow progress of this event, the previously formed adhesions usually prevent a general peritonitis, and merely a perigastritis occurs. Perforation is, however, an uncommon event, much more so than in simple ulcer, perhaps occurring in 4 to 10 per cent. of cases. Combined statistics show 4.46 per cent. (43 out of 963 cases). In this way abnormal communications form between the stomach and other viscera and tissues, *e. g.*, large intestine (gastrocolic fistula in 25 of 877 cases, *i. e.*, 2.2 per cent.), small intestines, the anterior wall (Mislowitz found 17 cases, Brinton only 1 in 507 cases), pleura, and pericardium, with admission of air and infected material into these cavities, the lungs and bronchi, with formation of abscesses, gangrene, etc.

Sometimes in the stomach there is partial healing; scars form, although the process does not cease, and in other parts the neoplasm shows progressive degeneration. Ulceration occurs in a very large number of cases, probably in more than 80 per cent., and hemorrhages are a common result, the amount depending on the size of the eroded vessel or the degree of capillary oozing. Suppuration occurred in 4 out of 40 cases observed by Boas.

Extension outside of the stomach occurs through continuity of structure, contiguity by means of adhesions, or by direct implantation, chiefly, however, by the lymphatics and sometimes by the blood stream, especially the portal vein.

Metastases originate either by the blood stream or lymphatics. They may appear before any suspicion is aroused of the gastric origin of the neoplasm. Thus, Tilling, in 1899, described an instance in which a secondary node on the humerus was the first evidence of the cancer whose origin lay in the stomach. Usually, however, the growths in the liver or peritoneum mask the original site.

Metastases travelling by the *blood stream* most commonly enter the portal vein and liver. They also commonly extend to the peritoneum and omentum, the pancreas, spleen, kidneys, pelvis, bones, lungs (especially the left), and brain, in such a manner as to suggest only one way of extension, *viz.*, through the circulatory system. No organ is immune.

The *lymphatics* allow metastases to form most commonly in the neighboring glands (85 per cent. according to Cuneo), chiefly those at the diaphragm, the perigastric and retroperitoneal glands, the extension occurring in the glands of the thorax and supraclavicular region as well as in the thoracic duct. Out of 556 cases the cervical glands were involved in only 21 (4 per cent.). Combined statistics show that in 2156 cases, metastases formed according to the following proportions in various parts of the body: glands, 44.1 per cent.; liver, 33.2 per cent.; peritoneum and intestine, 27.6 per cent.; pleura and lung, 7.3 per cent.; and pancreas, 7.6 per cent. In the Tübingen statistics (1891 to 1905) the distant glands were involved only in 7 out of 91 cases; in only 3 were the supraclavicular glands involved, and in the others the bronchial and mediastinal glands.

Cancers, of course, extend always to a greater or less degree beyond the area of induration, and evidence can only be found on microscopic examination of the surrounding tissues, where isolated groups of cells from the neo-

plasm may be seen, separated by areas of healthy tissues from the original source. Cancer of the cardia often extends to the œsophagus; that of the pylorus, on the other hand, rarely involves the duodenum.

The abdominal wall is at times invaded, chiefly through lymphatics of the obliterated umbilical cord, and great diagnostic importance attaches to the discovery of small secondary nodules in the subcutaneous abdominal tissue, especially about the umbilicus.

Peritoneal cancer is common, and there may be ascites from a variety of causes, among others from portal obstruction through pressure of infiltrated glands on the vein, or by direct invasion into it. The ascitic fluid may be serous, hemorrhagic, or chylous.

The shape of the stomach varies according to the situation, extent, and secondary effects of the neoplasm, and also according to its special character. Gastrectasis occurs with pyloric tumors, while if the cancer be at the cardia the organ may even be smaller than normal, as will also occur with cirrhotic infiltrating cancers in many cases. All forms of abnormality in shape will be found in cases where cicatrization and adhesions exist to distort the normal outlines. The stomach is often lower in the abdominal cavity than is normal, and very often it assumes the vertical position.

Secondary gastric cancer sometimes results from a primary growth in the stomach, *e. g.*, a cancer of the cardia may follow upon a pyloric growth, or, again, a pyloric stenosis may occur from compression by periportal cancerous glands, which become involved from a primary cancer of the cardiac orifice.

The general morbid anatomy reveals, as in any cancer, marasmus, with brown atrophy of the heart and fatty degeneration of its muscle, brown atrophy of the liver, fatty kidneys, and not uncommonly degenerations in the spinal cord and peripheral nerves. Multiple thrombi associated with the marasmus or arising directly from the neoplasm are sometimes found in the bloodvessels. General anasarca, anæmia, and cachexia are superadded to the picture.

Symptoms.—The symptoms vary according to the stage of the disease, the situation, the extent and pathological character of the growth, and upon its direction of progression.

Latency.—The onset may be quite unnoticed either by patient or physician (*i. e.*, no subjective or objective signs may exist). It is sometimes an accidental discovery at autopsy. Patients with gastric cancer may enjoy apparent health for months, with undisturbed appetite and digestion, and with maintenance of strength and nutrition even when extensive metastases are present, as described by Cheney and others. It is those cases occurring elsewhere than at the orifices that are especially apt to be latent. Absence of tumor and gastric symptoms is common in such patients, and the general picture of marasmus, or “primary liver cancer,” is apt to obscure or conceal the original location of the disease.

Latency is very apt, too, to occur in cancers of the cardia when no obstruction has yet occurred, and when from its position no tumor can be felt. Sometimes these cases simulate pernicious anæmia. The examination for occult hemorrhages in the stools, which is now a routine method, has done much to clear up cancers of the digestive tract which have hitherto been obscure by other methods of examination.

Onset.—Symptoms usually begin with inexplicable “dyspepsia,” which does not respond to treatment. Following upon this are pain, loss of weight, and

weakness, later vomiting, perhaps of blood, and the cachexia progresses to a fatal issue. The onset and persistence of dyspepsia in a man over forty years of age, who has previously enjoyed good health, is a suspicious circumstance. Sometimes the onset is apparently sudden, in the midst of health or after some acute illness, or perhaps after a trauma. Hammerschlag has already drawn attention to the frequency with which gastric cancer may thus develop. This "sudden" onset followed by progressive loss of flesh and weakness is very significant of cancer. In one of the writer's cases, the patient, a woman, developed her first subjective sign of indisposition five weeks before her death, the earliest symptom being a sudden pain in the right side of the abdomen coming on while she was hard at work and in apparently perfect health. Rapid weakness followed, and three weeks later jaundice set in, with progressive emaciation. Vomiting occurred one week later, a few days prior to her death. The examination on admission revealed a huge mass, probably of cancerous nature, and the autopsy confirmed the diagnosis, the primary growth being at the pylorus, where it formed a valve-like opening which permitted transmission of food in the proper direction. The liver was almost entirely replaced by metastatic growths, and all this with symptoms of less than five weeks' duration.

In one class of cases the onset is often characteristic, viz., where *cancer follows ulcer* or is engrafted upon it. In many of these the diagnosis is obscure through the presence of hyperacidity, especially as the HCl may be in excess to the end, and with it hypersecretion and early motor insufficiency; perhaps all the more so because spasm develops at the pylorus with the hyperchlorhydria. The deficiency of HCl in these cases appears late, and lactic acid is correspondingly small in amount and only found late. Pain and hemorrhages are early signs in these cases and usually marked features.

Early Digestive Symptoms.—*Flatulence* is frequent and the gas expelled is at first odorless, because H_2S is not usually generated in these cases. Later on with degeneration or ulceration of the neoplasm, the expelled gas has a foetid or foul odor from gangrene, suppuration, or the presence of organic acids. *A sensation of fulness* occurs even after very little food.

Anorexia is common (50 to 80 per cent. of cases) and early, and is apt to be progressive, although in many cases (one third, Boas) the appetite is preserved until late in the disease. Again, it may be perverted and capricious, and an especial dislike for meat may develop. There is no definite relationship between appetite and motor function or secretion. The taste is altered and often perverted. Smokers commonly lose all desire for tobacco. *Bulimia* has been described by Hanot in one case, but is very rare. *Dysphagia* is rarely experienced (except in cancer of the cardia), and was present in 10 out of 306 cases (Perry and Shaw), depending, of course, on the seat, development, and metastases. *Thirst* is definitely related to the degree of stenosis and to the motor insufficiency. It is thus more constant in pyloric cancer and less when the curvatures are involved. *The tongue* is usually thickly coated, although at times clean for months. Much depends on the condition of the teeth, gums, and pharynx.

Vomiting.—This is very frequent, and occurs in 80 to 90 per cent. of all cases. It usually takes place late in the course of the malady, and is especially common in pyloric cancer, owing, of course, to the stagnation and dilatation. In Warren Lyman's statistics from the Royal Victoria Hospital, it was noted in 68.2 per cent. Sometimes it is early (in 7 out of 83 of our

patients it was the first symptom, and in 34 it was very early); when this is the case, it is usually at longer intervals, and increases in frequency as the disease progresses. It is also frequent, and appears early when the stomach cavity is small from infiltration of the neoplasm. As dilatation becomes more pronounced the vomiting may again be at longer intervals and then more copious, the food being largely unaltered and coarse, mixed with mucus, blood pigment, detritus, etc., and having a foul or fœtid odor from organic acids. When an ulcerating obstructive growth at the pylorus breaks down to create a new passage, the vomiting may cease for weeks and the motor insufficiency be temporarily relieved. Acute fatal cases with rapid emaciation and repeated uncontrollable vomiting have been described. Vomiting is much less frequent when the fundus or anterior wall is the original seat of the cancer. Absence of vomiting does not depend on ulceration. In 26 of our 83 cases, vomiting was either absent until death or until discharge from the hospital. When the cardia is affected alone, vomiting will be more of the nature of a regurgitation and with much mucus. It is, as a rule, preceded by nausea, salivation, and digestive discomfort.

Effect of Vomiting.—As a rule, vomiting causes some relief to the general discomfort, but not much to the pain. In 14 of our patients the pain was relieved, and chiefly in the pyloric cases.

The time of appearance of vomiting varies with the site and nature of the growth, the degree of mechanical and functional disturbance, and to some extent with the nature of the food ingested. Late in the disease it appears one or more hours after a meal, and may be periodical, every two or three days, or may come on quite irregularly. Often three layers form in the vomitus where the retention of food has existed, the uppermost frothy, the middle layer consisting of turbid fluid and partly digested foodstuffs, the bottom layer being granular and containing food in various stages of digestion and other matter foreign to normal digestion.

Hæmatemesis.—Hæmatemesis is a common symptom, but occurs as an obvious sign in less than half of all gastric cancers. Chemical tests for occult blood, however, show it to be much more frequent. It is important to distinguish between true coffee-ground vomiting, and altered foodstuffs which have a similar appearance. This can be determined often only by chemical tests, by the microscope or the spectroscope.

The bleeding may occur from erosion of a large vessel, and be copious and even fatal, although this is rare (1 per cent. Welch); death may occur from hemorrhage without emesis. More usually it consists of small parenchymatous hemorrhages, either from the degenerated tumor, or from superficial erosion of the mucosa. The amount of blood is usually small, unless cancer is grafted on an ulcer, but may be oft repeated and oozes slowly, thus remaining a long time in the stomach. For this reason it is altered to hæmatin, giving to the contents the coffee-ground appearance. Tiny specks of dark clotted blood, which frequently appear in the vomitus with cancer of the lesser curvature, are of great diagnostic importance, and probably come from small hemorrhages in which the blood remains in folds of the mucous membrane or in depressions of the neoplasm.

This is often a very early sign of cancer, especially if associated with the Oppler-Boas bacillus, and the specks are often overlooked because, being of an intense black color and very small, they are mistaken for other things. They are most common in incipient cancer, especially of the lesser curvature.

The blood, although sometimes pure, is usually mixed with food. Repeated hemorrhages of small amounts is an important diagnostic point, especially if accompanied by diminution of HCl and the presence of lactic acid.

Occult bleedings are a very important finding, and, as will be seen later, form one of the most constant signs in the disease. (See the section on occult bleedings, under gastric ulcer.)

Pus is less commonly vomited, but when present may usually be told by the naked eye, or by its foetid odor. If in small amount it may only be revealed by the microscope. It arises through degeneration and ulceration of the neoplasm, and its presence is important for the diagnosis, it being rare in other chronic gastric affections. Mucopus, if present, is less foetid.

Pain.—Pain is usually preceded for a time by a mere sense of pressure and discomfort, especially after meals. It was the first symptom in 25 out of our 83 cases in the Royal Victoria Hospital (31 per cent.), and was present in the gastric region in 75 per cent. Later on it is usually although not necessarily present, and occurs in from 75 to 90 per cent. at some time during the progress of the disease. In Osler's series 13 per cent. ran a painless course. In the Royal Victoria Hospital, 22 per cent. had no gastric pain (Lyman). The time of its appearance varies within wide limits, sometimes occurring only every few days or weeks, sometimes only at night, etc., while at other times it is constant.

The degree of intensity, as a rule, is not excessive. At all events, it is not usually so severe as in ulcer. There is painful distress or a dull ache, usually fairly well localized to the gastric area. Sometimes it radiates to the back and to the sacrum. If the cancer be at the cardiac end, the pain may be at the lower end of the sternum. There is nothing characteristic about the pain. In pyloric stenosis peristaltic, cramp-like pains occur, and there is a sense of unrest.

A relation between the presence of a tumor and the site of the pain does not usually exist, nor between the absence of pain and the site or nature of the tumor. In some of our cases with diffuse infiltration of the stomach no pain existed, and pain was absent in at least one of each typical variety of regional disease. Cardiac cancer was especially free from pain. A relation to food may or may not exist. Often food increases the pain, especially late in the digestive period (if the cancer be pyloric), or the pain may be continuous and bear no relation to the ingestion of food.

The Bowels.—The bowels are rarely regular, perhaps in 5 per cent. of cases. They are usually at first constipated. Diarrhœa occurs less often—F. Müller says that diarrhœa occurs in 35 per cent. of all gastric cancers—and at times the two conditions alternate. There is no relation between the growth itself and the condition of the bowels, except that when retention of food occurs through pyloric stenosis, constipation is more apt to exist.

Blood in the stools, usually as occult bleedings, is present in a very large percentage of cases, probably in over 90 per cent., and this finding is a valuable adjunct to the diagnosis. It may be obvious or merely be detected by chemical methods.

The Urine.—The quantity is usually diminished in amount and concentrated—according to the degree of gastrectasis and pyloric stenosis. The *chlorides*, while often less than in the normal, may vary within wide limits, for the retention of chlorides in the organism depends on other factors than the specific action of the carcinoma. *Phosphates* are increased in quantity

on account of insufficient food, but not through the action in any way of the cancerous process. *Nitrogen* excretion in the urine is usually greater than normal, the increase depending not alone on the food (for it cannot be raised with additional food), but on the great albumin destruction that occurs in gastric cancer. So great is this destruction that Salomon has attempted to utilize it for diagnosis by testing the lavage water quantitatively for nitrogen and albumin to gain an approximate estimate of the presence of gastric malignant disease.

Albumin is frequently present (perhaps in one-fourth of all cases), although usually in small amount. Large quantities of albumin are exceptional. *Albumoses* (peptonuria) have been found from time to time in gastric carcinoma. A certain diagnostic significance attaches to their presence, as indicating the possibility of malignant disease and the probability of its ulceration. Aldor examined 56 unselected cases, and found albuminuria in every patient with fever, and in 5 others in whom cancer of the stomach was present, and in one with cancer of the omentum. It is merely a confirmative test.

Acetone and diacetic acid are sometimes present, but usually only in the last stages of gastric cancer, and are of no diagnostic import, occurring as they do in other forms of gastric disease in which inanition, toxic states, etc., may exist (gastrectasis, ulcer, etc.). *Indican* has no pathognomonic significance, being common in the urine in numerous derangements of the alimentary canal, and being variable in amount even in the same diseases. Urobilin, when present in excess, does not arise from the cancer, but from some complications, *e. g.*, hepatic disease, pneumonia, etc. *Ethereal sulphates* are increased in the urine only where HCl is deficient, but not necessarily so, even under these conditions, so that in carcinoma their occurrence is most variable. *Volatile fatty acids* are in excess sometimes when cancer occurs with stasis and diminished HCl, but Sigel doubts the diagnostic value of this finding. The variations of these acids is too great to be relied on (Rosenfeld).

The presence of *sugar* in the urine implies the probable involvement of the pancreas or brain and perhaps of the liver.

Nutrition.—Emaciation is the rule; it is usually progressive, and is often the first symptom to arouse suspicion. The skin becomes lax, dry, and wrinkled, the color earthy, sallow, and sometimes jaundiced. Cachexia supervenes, especially with motor insufficiency. There is nothing pathognomonic about the cachexia, and it has no diagnostic value per se. Two exceptions are worthy of mention: (1) Sometimes patients run an advanced course with well-maintained nutrition, for this is by no means incompatible with gastric cancer. (2) Remissions occur, patients regaining some or much of their lost weight, especially under careful lavage and diet with encouragement, although such an improvement is necessarily of short duration. In cancer of the lesser curvature, there is usually but little emaciation early in the disease.

Loss of Strength.—Loss of strength usually accompanies emaciation and is an important factor in the diagnosis, although at times the strength is well maintained until late in the disease.

Anæmia.—Anæmia is due to the toxæmia, hemorrhage, and malnutrition. The blood examination is of little value in the diagnosis. Usually there is moderate leukocytosis. The red cells are normal or much diminished, sometimes there is marked poikilocytosis, and the picture may resemble that

of pernicious anæmia. The hæmoglobin is normal or much diminished. The average red count is usually above 2,000,000 per cmm.; the average hæmoglobin is about 50 per cent. The absence of digestive leukocytosis has no practical diagnostic value. In the first place, it varies greatly in cancer, while, on the other hand, it is not uncommon in benign pyloric stenosis.

The specific gravity and density of blood are of no value except as aids to diagnosis in a general way. The specific gravity goes hand in hand with the amount of hæmoglobin, and hence is often diminished.

Fever.—Fever occurs in about one-half or three-quarters of all cases, and at varying and irregular intervals. Sometimes there is an initial fever, or again during the course of the malady we may get intermittent types or irregular temperature, and not uncommonly, especially with marked degeneration and ulceration of the neoplasm, sudden elevations to 105° or 106° occur with rigors and as sudden a termination. At other times the type resembles that seen in malaria with chills and sweats. These irregularities in temperature are probably toxic, arising from absorption, although at other times the fever is due to complications, *e. g.*, peritonitis, pleurisy, etc., which should be looked for. Terminal pneumonia or pulmonary metastases often cause the fever which occurs during the latter days of the disease.

The neck may show enlarged supraclavicular lymph glands, especially on the left side. They become swollen, palpable, perhaps visible above the clavicle. The diagnostic value of this is doubtful. Associated with invasion of the thoracic duct, their presence was regarded as an important sign. Yet this may occur in all or any visceral cancer. Again, their appearance is usually a late sign, not an early one. Sometimes, however, it is early enough to be of use, and therefore should be looked for. Hechler found 18 cases in 70, Lepine 3 in 40. In the Tübingen statistics (1891 to 1905) only 3 cases out of 91 autopsies on gastric cancers showed involvement of the supraclavicular glands.

The Abdominal Examination.—Inspection.—Inspection is of great importance. Osler has drawn special attention to examination of the abdomen for unevenness of the furrows below the ribs, for fulness in the epigastrium, nodules in the skin and about the navel, peristalsis and anti-peristalsis, and, lastly, for a wide area of aortic pulsation. From inspection one may often see not only a tumor, but gastroptosis and gastrectasis. It is well to inspect the recumbent patient from behind the head of the bed. Cancer of the lesser curvature is often easily seen as it descends with deep inspiration, and one may make out its sausage-like shape and see it ascend with expiration, especially when the stomach is empty. With a full stomach a tumor may be pushed upward beneath the ribs or liver and be invisible.

The mobility and movability of the tumor vary greatly. The mobility may vary with the position of the body, with the respiratory movements, with inflation of the stomach, and with palpation. It is detected chiefly by inspection, palpation, and inflation, and much depends on the presence or absence of adhesions. Various positions of the body may coincide with altered positions of the neoplasm, and some large and localized growths may thus readily change their site from time to time.

Respiration often has a marked effect on the position of the tumor, and if it be localized to the pylorus, circumscribed and solid, and non-adherent to surrounding viscera, it may alter greatly, descending with inspiration and

returning with expiration. It may be accepted as a general rule that absence of respiratory mobility implies non-adherence to the liver.

Cancers of the curvatures move readily, and may show fixability on expiration. This aids in the differentiation both of cancer of the stomach from that of other organs, and of cancer of the pylorus from that of the lesser curvature. Often tumors which descend with inspiration may be held in that position during expiration.

Absence of this expiratory fixability does not necessarily prove adhesions to the liver or diaphragm, and therefore, if the tumor move with the liver, it is not necessarily adherent to it, but such adhesions may usually be excluded if the tumor on expiration acts differently from the liver itself. Even this is not an infallible rule, however. The changes of position depend on the size, the site, and the extent of the tumor. A large, wide tumor, adherent to the liver and yet extending below it, may be partly fixed even when the liver returns with expiration. On palpation, these tumors are sometimes found quite movable, especially if non-adherent elsewhere and if confined to the pylorus. Sometimes they may be moved to almost any position in the abdomen, as in one instance in a woman admitted with localized cancer of the pylorus with extreme gastrectasis, evidently a carcinoma developing over a previous ulcer. The tumor could at will be pushed beneath the right costal margin, to the right iliac region, or across the epigastrium to the left of the median line, and even below the umbilicus.

Insufflation and inflation have some influence on the situation of a neoplasm in the stomach. *Inflation* may be carried out by administration of separate powders of tartaric acid and sodium bicarbonate, or by insufflation of air through the stomach tube by means of a Davidson syringe. One should be careful in this method not to inflate to excess, if one would avoid injuring the mucosa. The writer saw a severe laceration of the mucosa under such conditions in a European clinic, where the autopsy revealed four or five linear tears from end to end of the organ.

Pyloric tumors descend to the right, and rarely ascend to the right. Tumors of the lesser curvature move backward and disappear because the larger curvature turns forward, just as happens with a full stomach. By inflation, too, the position and size of the stomach are easily detected, and ptosis becomes more obvious, especially in cancer of the lesser curvature. It is important to remember that in pyloric cancers there is not always gastrectasis; and that gastrectasis does not necessarily exist because stasis is present. The stomach may remain normal or even get smaller, *e. g.*, with diffuse infiltration or cardiac cancer.

Palpation.—This is the best and most certain of all the methods to tell the presence of a tumor. The examination should be made in various positions, and if necessary in a hot bath, or under ether, in order to determine the size, shape, situation, mobility, character, etc., of the growth. In the absence of a palpable tumor, the diagnosis often remains dubious. Sometimes a tumor is not accessible to palpation even when large, especially if (1) it is high up in the cardia, (2) if it is in the lesser curvature and fixed to that vicinity, or under the left lobe of the liver, or (3) if it is in the posterior wall of the stomach.

Tenderness of tumors is common, in variable sites, and there are often tender points behind, near the vertebræ.

It is often possible to feel a hardness and the nodular character of a

neoplasm and the knobs of cancerous masses with central depressions of degeneration (Farre's tubercles).

Pyloric tumors are the easiest of all to palpate, especially in women with lax abdomens and descended stomachs. Not infrequently one may feel the gurgling of gas through the pylorus.

Gastric Rigidity.—Boas has drawn special attention to this symptom, although Cruveilhier noted it more than half a century ago. As Boas has observed, the rigidity may exist merely as slight contractions of short duration confined more or less to the fundus, or as more marked tonic spasms of the muscle over a larger area, causing prominences of the fundus, and creating a sensation of pain to the patient, or, again, there may be still more decided contractions, which are both visible and palpable and are of longer duration. Gastric rigidity implies pyloric obstruction, and it is best observed with a full stomach.

Percussion.—Dulness or dull tympany may exist over a large tumor. There is increased tympany over gastrectasis. Inflation aids this outlining.

Auscultation.—Auscultation and auscultatory percussion are of doubtful value, although perhaps future developments may demonstrate their usefulness. The sign of the deglutition sounds, viz., delay in their occurrence and transmission to the ear, has a doubtful value, and is certainly not pathognomonic. It is possible to hear, too, the gurgling of gas through the pylorus. Friction from a localized peritonitis is, of course, sometimes audible.

The Functional Signs and Analysis of the Stomach Contents.—There is no one pathognomonic sign of cancer. The diagnosis is made from the symptom and sign complex. The signs vary greatly according to the site, size and extent, shape, character, etc., of the neoplasm. Pyloric cancer gives a different picture from that of cancer in the lesser curvature.

The macroscopic evidences in the stomach contents show chiefly poor proteid digestion, while the carbohydrates have been well acted upon. If pyloric obstruction or general weakening of the muscle wall of the organ has occurred, the amount retained is abnormally great. Tumor fragments are rarely seen. Blood is common, and sometimes there is pus.

The *motor power* is nearly always diminished in all forms of gastric cancer. It is not necessarily associated with dilatation, for a small, rigid, cancerous stomach may equally well show motor insufficiency. Often the motor insufficiency is only of the first degree for some time, but this in itself is an important feature for diagnosis, because motor insufficiency usually appears as an early sign, more especially in pyloric cancer, and before the tumor is observable. It rapidly becomes more severe, especially with marked stenosis and insufficient hypertrophy of the muscularis. It may be slight at first. In cardiac cancer it is less frequent than in cancer in other situations, although even here it comes with infiltration and extension to other parts, or with a concomitant catarrh.

In cancer of the lesser curvature, motor insufficiency is slight at first until extension and infiltration have occurred, involving the pylorus, but not necessarily causing stenosis so much as preventing peristalsis and inducing a rigidity of the wall and hence motor insufficiency with insufficient pylorus. In cancer of the body of the stomach, more than 50 per cent. of cases show motor insufficiency (Boas 64 per cent. in curvatures and fundus), because of the infiltration of the walls and their weakness through toxæmia and cachexia.

Where *marked motor insufficiency* occurs, food may be retained for days

or even weeks and the increase is usually progressive; when pyloric stenosis exists the advance is often rapid. Stagnation of solids is especially noticeable in cases without stenosis.

Secretory Power.—There is usually a gradual and progressive diminution of the free HCl until it is absent, and the ferments also disappear in part or entirely. This occurs in perhaps 80 to 90 per cent. The presence of HCl is no proof of the absence of cancer of the stomach. One examination of the contents is never enough to form conclusions. It is the persistent diminution or absence of HCl which is significant. Variability in the amount of HCl secreted in carcinoma ventriculi is probably much greater than is commonly recognized, and the total acidity varies within still wider limits. Combined HCl is often absent.

Gluzinski has drawn attention to the importance of a diminution of HCl in diagnosis. By his method three tests are given in one day: (1) On an empty stomach. (2) After an albumin meal (two hard-boiled egg-whites and 100 to 200 cc. water). (3) After a test dinner. One should find HCl absent in two out of these three tests if cancer be present.

The theory of this test is based on the fact that as a result of acid catarrh a chronic mucous gastritis occurs. This is present under two important conditions, viz., where an ulcer heals without the formation of a stenosis, or where an ulcer becomes cancerous. If one gets a weak reaction at the first test, and at the other two a marked reaction, it implies the commencing change from acid catarrh to gastritis. In pure ulcer we get a reaction after all three tests. If, however, cancer is developing on the ulcer, we have progressive secretory insufficiency.

Still, perhaps the disturbed secretion is due less to a gastritis than to the fact that the HCl, although secreted, is quickly taken up by products resulting from the cancer. Emerson found that in ulcerating cancers certain basic substances were produced by autolytic processes, which combined with HCl. He believes these to be "hexone bases, the result of the digestion of the proteid by a ferment furnished by the tumor itself." With cancerous dilatation we may get free HCl in the early morning, but not one to two hours after breakfast or four hours after dinner. The important feature is the early disappearance of HCl with normal total acidity; later in the disease the total amount also falls off. Sometimes it disappears suddenly and may return after the cancer has been extirpated. As a rule, in pyloric cancer there is a normal amount of HCl, persisting longer than in the other forms (even without previous ulcer), because the cancer usually remains circumscribed for a longer time. Not infrequently we get excess of HCl with or without ulcer.

Ziegler, Sailer, and others have reported cases of hyperacidity with cancer of the stomach occurring early in the disease. The writer has observed repeatedly such cases in which the early hyperacidity lasted for more than a year before cancer was at all evident. In one of these cases the autopsy showed no sign of previous ulcer, but a carcinoma *en plaque*. Ziegler distinguishes three stages of hyperacidity in cancer without previous ulcer: (1) Uncomplicated hyperacidity in old people, formerly well. (2) Hyperacidity, but no lactic acid and no bacilli, like the condition in simple ulcer or in mere hyperchlorhydria, but treatment is of no avail. (3) Gastric analysis shows normal HCl or subacidity, with some mucus and some motor insufficiency accompanying a declining health.

In cancer of the fundus we get absence of HCl much earlier, because the process is more diffuse, and therefore it affects the secreting glands more. In all cases one must distinguish between hyperchlorhydria and mere hyperacidity, which is often due to organic acids as well. Hydrochloric acid may be present throughout the whole course of the disease. The predisposing conditions, therefore, are a localized tumor, especially if pyloric, and secondly cancer developing on an ulcer.

Absence of HCl is but one sign, and is of partial importance only; for it occurs, too, in gastric neuroses, catarrh, atrophy, etc., and in fevers, constitutional disturbances, cardiac disease, etc.

Organic Acids: Lactic, Butyric, Acetic.—The presence of *lactic acid* depends mainly on diminished motor power and lessened HCl, and therefore lessened digestion of albuminous foods, *i. e.*, lessened ferment activity because of atrophy of the glands. Sometimes it is present in small amounts, even with good motor power, through retention of bits of foodstuffs in folds of the mucous membrane. There must be little or no free HCl, although the combined acid may be abundant. It may be introduced preformed into the stomach by the food, *e. g.*, bread, milk, fish, meat, etc., and for this reason Boas recommends a test meal of oatmeal and water. Still there is scarcely enough lactic acid in the food of an ordinary test breakfast to signify. He gives his meal at night into the previously washed-out stomach, and removes it in the early morning.

Lactic acid is formed in the stomach, however, only under abnormal conditions, and is the commonest organic acid found in cancer. In 30 to 80 per cent. of gastric diseases, with the presence of lactic acid, there is lessened albuminous digestion. The origin of it may be from acid producing microorganisms in the stomach, *e. g.*, the Boas-Oppler bacillus, or from some special ferment associated with the cancerous tumor. Lactic acid is not pathognomonic of cancer, and may be present in intestinal obstruction, sometimes even in benign pyloric stenosis, in severe chronic gastritis, in atrophy of the mucous membrane, fat necrosis of the pancreas, cardiovascular disease, gall-bladder cancer, pernicious anæmia, etc. Nevertheless, its *early appearance* in cancer of the stomach, together with a *diminution of the total acidity*, even before any signs of stenosis, is very important.

Very little HCl, free or combined, will suffice to prevent lactic acid fermentation, unless there be marked atony. Therefore the fact that HCl is not uncommonly present in cancer of the stomach reduces the value of a negative test for lactic acid. In other words, its absence is no proof that cancer is not present. Still, as Boas shows from combined statistics, 84.4 per cent. of all gastric diseases in which lactic acid is found are cancerous, and Schiff's combined statistics show that 73.5 per cent. of gastric cancers give positive tests for lactic acid.

Does lactic acid usually appear early, *i. e.*, long before the gastrectasis? Kuttner and Lindner say it does not appear before a tumor is felt, but this is, of course, too general a statement. The time of its occurrence varies. Usually it is not an early sign, because it depends in part on atrophy of the glands and motor insufficiency. Still, these *do* sometimes atrophy *early*, as Hammerschlag and others have shown. The mucous membrane is sometimes destroyed even when a tumor is quite localized, and, vice versa, with extensive cancerous disease the mucous membrane is sometimes preserved. Hence the variations in time of the appearance of the lactic acid,

Its constancy is very suggestive, for the persistent presence of lactic acid is rare in gastric diseases other than cancer.

Ferments.—Hammerschlag, in 1884, examined 280 patients with gastric disease, and found the peptonizing power (according to his method) below 15 per cent. in only 32 cases, of which 26 were cancers. In these there was no albuminous digestion in 23, and in the other 3 it was below 15 per cent.

Out of his 280 cases, 42 were cancerous; therefore 26 out of 42 show this extreme lack of the fermenting process. Oppler denies this on the basis of an examination of 26 cases, in which he found merely that HCl secretion and ferment activity ran parallel, indicating that diminution of the pepsin cannot be regarded as an early sign of cancer. It is, moreover, not always present in cancer, and, vice versa, is present sometimes in benign stenosis with gastrectasis and in neuroses.

The presence or absence of secondary atrophy of the glands of the mucous membrane is alone the decisive feature in the production of ferments. Early diminution of ferment production, even when HCl secretion is preserved, has been repeatedly observed.

Occult bleedings from the stomach or bowel should be carefully looked for as being one of the early and most important signs of gastric cancer. The Benzidin test should be used. The importance of this sign in the diagnosis depends, however, on their persistent presence and on their association with gastric subacidity. If occult bleedings occur in the stools, when HCl is present in the gastric contents, the diagnostic value is less, as ulcer could also produce similar results. If occult bleedings are not found in the feces, and there is gastric subacidity and good motor power, it argues against the presence of cancer.

Microscopic Examination of the Stomach Contents.—This is more or less valuable according to the period of the disease. Blood cells and pigment are commonly found. Pus cells vary in amount, and at least a few leukocytes can almost always be seen. The presence of blood or pus or both in the early morning lavage is of great significance.

Bits of mucous membrane and of cancer tissue are not commonly found. A few cases are recorded, but being a late sign, this has little value. The lavage water frequently has little shreds of torn mucous membrane, because the mucous membrane in cancer is friable. Examination of these shows widening of the submucous tissue at expense of the true gland layer, *i. e.*, glandular atrophy. It is often hard to be sure of this, however, because it depends upon which part of the mucosa accidentally comes away in washings. Often mere catarrh is evident, and this is not specific for cancer. Sahli, however, laid great stress on this, believing that a thorough lavage at night, and again of the empty stomach in the morning, would reveal in the last washing torn portions of the tumor. A true bit of tumor is the only single pathognomonic sign of cancer of the stomach. But this means ulceration, and therefore a late stage in the disease. Lubarsch regards early diagnosis by this means as impossible. Malkow found bits of tumor in the feces.

Boas-Oppler Bacilli.—These have been frequently found in the stomach contents in gastric cancer. They have been cultivated by Schlesinger and Kaufmann in pure culture on sugar agar, on beef peptone agar with the addition of cancer juice, and on grape sugar agar, although best of all are media to which blood is added. Hence they are frequent in coffee-ground contents. The average length of the bacillus is 6 to 8 μ , although it varies between

3 and 10 μ ; sometimes it occurs in long spirals and continuous threads, 1 μ broad. It is non-motile, Gram positive, stains a deep blue-red color with aqueous methylene blue, is facultative aërobic, and has no spores. It coagulates milk.

Schlesinger and Kaufmann demonstrated the formation of lactic acid from various forms of sugars by these bacilli, therefore they may be regarded as one of the excitors of lactic acid formation in the stomach, but only one; there are others, and therefore they are not specific for lactic acid formation. They have been found in 22 out of 70 cases of gastric disease; of these 22, 19 were cancers and 3 presented other conditions. Their absence does not prove the non-existence of cancer. Heichelheim emphasizes the findings in gastric contents of clots of blood with these bacilli, in the absence of HCl, as almost pathognomonic of cancer.

Ziegler has recently called attention to the microscopic evidence of stasis as important in the early diagnosis of cancer, especially the finding of the Boas-Oppler bacilli amid mucus which may plug the introduced tube, also food remains and heaps of leukocytes. These early signs he attributes to cancerous invasion of the lesser curvature, hence the rigidity, the lessened peristalsis, and slight stasis.

Sarcinæ are uncommon unless free HCl is present; therefore, as a rule, they are only present when the cancer has formed on an old ulcer. The presence of *sarcinæ* is no proof that cancer is not present, although if they are in abundance the diagnostic value is of some importance as suggesting a benign rather than a malignant condition.

A few years ago Cohnheim recorded six cases of cancer with *trichomonas* and *megastoma entericum*. He attaches diagnostic importance to their presence in early stages before the lactic acid forms. They cannot live where lactic acid exists, and are hence more likely to be found in cancer of the cardia and lesser curvature, for in these cases often the lactic acid develops late. Others partially confirm these findings (Heuser, Strube, Zabel).

Amœbæ and *flagellata* are found in cases of cancer of the stomach in which no stasis exists, but in which an alkaline reaction obtains on the gastric surface, *i. e.*, non-pyloric cases. Cohnheim thinks them an early sign, and that they are found before the ulcerating tumor has become gangrenous. Later they disappear.

Yeast cells sometimes occur. *Meat fibers* are found because of delayed and lessened proteid digestion. *Starchy foods* may be abundant, but are found to be more digested than the meats. *Fats* are likewise present.

Diagnosis.—Inasmuch as surgical intervention at the commencement of the disease is essential to a cure, the earliest possible diagnosis is necessary for rational treatment, and one must endeavor to determine not only the location of the disease, but the extent of its development, the presence or absence of complications, and the possibilities of satisfactory treatment. Where possible, a diagnosis should be established before a tumor is detected, although from recent surgical statistics this is not essential for a radical extirpation with cure.

Many tumors, even though palpable, are sufficiently localized to extirpate, and even when fairly large, a successful resection of the stomach has been accomplished. Then, too, many tumors feel larger than they really are, and vice versa. As regards palpation, it does not follow that because a

tumor is not yet palpable that the disease is in an early stage. Huge cancers with adhesions and metastases may have already formed and be entirely covered and inaccessible to palpation, and, vice versa, an advanced condition may remain localized for a long time and grow slowly long after the diagnosis is made.

The main evidence in the early cases lies in the findings of functional disorder. There is no single pathognomonic sign of cancer of the stomach, and one must consider the whole symptom complex, using for his purposes not only the history, but a careful examination of the symptoms, signs, the physical examination, the chemical and microscopic examination of gastric contents and stools, and such investigations require often prolonged, patient, and thorough observations. In spite of all there are numerous cases in which it is quite impossible to diagnose. One general rule holds in most cases, viz., *the absence of all gastric symptoms and signs is important evidence against cancer.*

Penzoldt has well said that in individuals over forty years of age with gastric trouble, one should not rest until one has satisfactorily decided for or against cancer. Klemperer advocates as a therapeutic test that elderly patients in doubtful early states should be placed on a rest cure with a protective diet for several weeks. If with this method improvement does not supervene, cancer may be strongly suspected.

General Signs.—When no tumor is evident one must rely on the age, family, and personal history, the history of the illness, especially its onset in the midst of good health, the anorexia especially for meats, the hæmatemesis, weakness, and emaciation in spite of apparently sufficient food, the motor insufficiency, and the results of chemical analysis and other investigations of the vomitus and contents from test meals, and upon the presence of persistent occult blood in the fæces. In the stomach contents, the cardinal features are evidences of motor insufficiency, the persistent diminution of HCl, the presence of lactic acid and of blood.

Tumor.—The possibility of palpating a growth depends much on the topographical conditions of the stomach and of the tumor, *e. g.*, cancers with adhesions, with extension upward of the growth, etc., may not be accessible to palpation. Where tumor is evident, one is called upon to decide (1) whether or not it be gastric, and (2) if gastric, whether it be of a cancerous nature or otherwise.

Certain *general tests* have been recommended as aids in the early diagnosis of gastric cancer before a tumor is palpable, although none of these have justified the claims originally made for them. The tryptophan reaction is based on the effect of chlorine or bromine to produce a red-violet color on the proteinochrom which forms as a result of the great albumin destruction. The test is not constant, having failed in 13 out of 15 cases (Sigel), and having been found positive in other conditions, *e. g.*, ulcer. Kuttner's opinion was similar to that of Sigel.

Salomon believed an earlier diagnosis possible by testing the lavage water for nitrogen and albumin, his test being based on the idea that in gastric cancer owing to the great destruction of tissue, an albuminous serum is poured into the stomach. The stomach is first carefully washed on the evening before testing, after a preliminary non-albuminous fluid diet for twenty-four hours. On the next morning the stomach is washed thoroughly with normal saline solution (400 cc.), the same fluid being re-

peatedly used and then tested by Esbach's and Kjeldahl's tests. More than 20 mg. of nitrogen to 100 cc. or 0.5 gm. of albumin, suggest the presence of cancer. In negative cases there should be little or no turbidity with Esbach's reagent, and the nitrogen by Kjeldahl's method is not greater than 15 mg. Minkowski is not in accord with these views. Gerster, however, regards this test as useful in cancer of the lesser curvature without stenosis, unless the cancer has formed on an old ulcer, in which case the little HCl present would digest the albumin present. Reicher, Sigel, Tabora, and Zirkelbach confirm the test as being at all events of some value, especially as indicating an ulceration of the gastro-intestinal tract, although not necessarily differentiating malignant from benign conditions. Furthermore, it may assist in diagnosing cancer from chronic gastritis unless the cancer be of an early diffusely infiltrating character.

Gluzinski's test for the relative diminution of HCl has already been mentioned.

O. Reissner's contention that the diagnosis of gastric cancer is aided by the determination of the chlorides in the stomach contents, is worthy of mention. In carcinoma ventriculi the chlorides are apparently much increased, despite the fact that HCl is itself deficient, the alkaline cancer juices evidently themselves containing the chloride excess. The method of Lütke Martius is employed, and while in other gastric diseases 100 cc. of gastric contents have a corresponding chloride value of 24 to 40 cc. of $\frac{N}{10}$ silver solution, in carcinoma 50 to 70 cc. $\frac{N}{10}$ silver solutions are required.

Differential Diagnosis.—Two main types of condition may be considered:

A. Gastric cancer without evidence of tumor.

B. Gastric cancer with evidence of tumor.

A. **Where no tumor can be detected**, one must take cognizance of two classes of cases:

I. Those with little or no motor insufficiency.

II. Those with definite gastrectasis.

A third class without tumor exists, namely, cancer at the cardiac orifice, which will be dealt with separately.

I. Where no motor insufficiency exists, the cases are easier to diagnose, because we can depend on the presence of lactic acid, which is rare in the other gastric diseases that have no atony. Therefore, if we happen to get lactic acid early, cancer is probably present. The main diseases in this group are:

(a) *Ulcer* without tumor or stenosis. Here the HCl and pepsin are usually normal or increased. No lactic acid exists, and there are other positive signs of ulcer, *e. g.*, the age and nutrition of the patient, the character of the pain and of the vomited matter, the course of the malady, effects of therapy, etc.

(b) *Neuroses*.—HCl and pepsin are usually normal in amount, although not always, and thus prolonged observation is necessary sometimes before deciding, especially in elderly people. Frequent analyses are essential, and the effects of therapy will materially aid the diagnosis. There are, moreover, no occult hemorrhages in the gastric contents or stools.

(c) *Chronic Gastritis*.—The history of a cause (alcohol), the gradual onset and slower progress, with remissions and exacerbations, are important features. The secretions are disturbed later in the disease, and the features are more local than constitutional. Hydrochloric acid is less constantly

absent and is often only diminished. Motor insufficiency is later too, progresses less rapidly, and there is less gastrectasis; there is also less stagnation of solids, and occult bleedings are less persistent, if at all present.

(d) *Atrophy of the mucous membrane* (including pernicious anæmia, primary atrophy of the gastric follicles, terminal stage of advanced chronic gastritis, cancer of distant organs, *c. g.*, breast, rectum, uterus, Addison's disease, etc.).

Primary atrophy runs a protracted course with gradual onset, and the motor power is unimpaired for a long time. There is no lactic acid, and little emaciation; anæmia, however, exists in this as in cancer. Advanced *gastritis* has a similar series of signs and symptoms, but some exceptions occur, and then the diagnosis is impossible. There is, however, no cachexia. Lactic acid is less constant, Salomon's test may be negative, and occult hemorrhages are far less constant. *Achylia gastrica*: There is no cachexia or emaciation, a fair general condition exists with good digestive power. Lactic acid is absent and there are no occult hemorrhages. *Pernicious anæmia* shows little emaciation, as a rule, although sometimes it is extreme. HCl may be absent in the contents, and sometimes even lactic acid is present. Even the blood counts may be hard to distinguish, but the cachexia and the anæmia do not go *pari passu*. There is no hæmatenesis, and occult bleedings are usually absent. Remissions and exacerbations are marked. *Addison's disease* resembles cancer because of the general malaise, weakness, dyspeptic signs, etc. But the pigmentary changes are present as a feature of the malady. *Cancer of distant organs* induces no motor insufficiency, and lactic acid is not present. In many cases, however, HCl is absent, as has been repeatedly observed in recent years.

II. *Cases with Gastrectasis*.—With gastrectasis and pyloric stenosis it is harder to differentiate, because the lactic acid is so often present in gastric diseases (other than cancer) with atony, and therefore its presence has less significance.

The main diseases to be differentiated are: *Scarred ulcer of the pylorus without palpable tumor* (which may lie beneath the liver or be too small for palpation). The history of previous ulcer, the longer duration of the malady, the presence of HCl (perhaps even in excess), the absence of lactic acid, the eructations of gas containing H_2S , and the findings of sarcinæ, all suggest the benign condition; and the absence of cachexia aids in excluding carcinoma. *Hypertrophic stenosis of the pylorus* (Boas' stenosing gastritis), if acquired, is usually due to ulcer and gives signs similar to the above. Sometimes a *carcinoma developing on an old ulcer* must be differentiated, and the diagnosis is not always easy; indeed, often it is quite impossible. As a rule, there is a history of old ulcer and the symptoms change in character, becoming more persistent, and resistant to treatment. There is, moreover, greater wasting, anæmia, and pain. Bleeding and perforations are not uncommon. HCl is present, often in excess, and there may be hypersecretion (continuous or alimentary). Gluzinski's tests are perhaps useful in these cases; at all events, if positive, they indicate the presence of cancer, and if negative, they do not prove its absence. Cancer developing on an ulcer shows a tumor more easily because of the usual perigastritis and pyloric spasm.

B. When Tumor Exists.—Two questions arise: (1) Is the growth gastric or extragastric? (2) If gastric, is it benign or malignant?

It is often well, as an aid to diagnosis, to palpate the abdomen with the patient in a warm bath or under ether, unless, indeed, an exploratory laparotomy be undertaken. Minkowski suggested inflation of the stomach, and at the same time distention of the colon with water, for differential diagnosis of gastric and perigastric tumors. With inflation of the stomach, pyloric tumors usually move downward and to the right; those of the lesser curvature disappearing, while those on the anterior wall and lesser curvature together become broader but less defined. Under similar conditions, tumors of the liver would move upward and to the right, those of the spleen, intestines, or omentum would move downward, while if the pancreas were the seat of the neoplasm, it would entirely disappear as the gas filled the distending stomach. Renal tumors would be unaffected by the inflation.

Inflation or distention of the colon with water would displace gastric tumors upward, as also those of the spleen. Tumors of the kidney disappear behind an inflated colon. Tumors of the liver would vary according to size and position, while those of the omentum would be displaced downward.

The consideration as to the location of the neoplasm in or outside of the stomach has already been dealt with in the discussion of the symptoms and physical signs.

Growths Located Outside the Stomach.—The tumors simulating gastric growths, but existing in reality outside the stomach, are mainly:

1. *Perigastritis* with an exudate and perhaps adhesions about the pylorus, or the lesser curvature, may induce chronic pain with dyspepsia and even dilatation, all simulating cancer. The main features against cancer are the long duration without sufficient corresponding emaciation and cachexia, and the different chemical signs on analysis. (See Complications of Gastric Ulcer.)

2. *Cancer of the Duodenum.*—This condition is rather rare. Schlesinger found only 7 primary cases in 25,000 histories. Rolleston could collect only 41 cases in the literature; of these, 8 were in the first portion, 5 in the first and second parts, 24 in the second portion, and 4 in the third portion of the duodenum; 10 were in females and 31 in males. Here the difficulties are great indeed; it may be impossible to diagnose the true condition. One may have diminution or absence of HCl. Lactic acid, on the other hand, may be present, as, indeed, may most of the other signs of gastric cancer.

3. *Omental or general peritoneal tumors* often cause difficulty in the differential diagnosis, especially if accompanied by ascites. Peritoneal cancer from any origin other than gastric may simulate cancer of the stomach, especially if in aged people with cachexia, and enlarged inguinal glands, pressure on the portal vein, and hemorrhagic ascitic fluid. But these neoplasms are less mobile with respiration, and usually give negative chemical tests on gastric analysis. The rectum should always be examined in doubtful cases of the kind.

4. *Peritoneal tuberculosis* with ascites may simulate gastric cancer with secondary peritoneal involvement. However, it occurs usually in younger subjects, runs a more protracted course, with exacerbations and remissions, and is accompanied by more persistent irregular fever. No occult bleedings occur. Dock has drawn attention to the value of cytological diagnosis of the fluid with its characteristic cells, in which mitoses are very common.

5. *Tumors of the transverse colon* usually give evidence of some degree of intestinal obstruction; the stools may be bloody. Examination of the stomach itself by test meals and inflation shows the absence of gastric disorder.

6. *Tumors of the gall-bladder* are situated in their appropriate place, are somewhat movable, and may by pressure upon the surrounding ducts cause early jaundice. At times, however, the differential diagnosis is extremely difficult. While there need be no motor insufficiency, yet adhesions in the neighborhood of the gall-bladder tumor may cause pyloric stenosis and certain signs of gastric cancer. The absence of respiratory mobility may suggest a gastric cancer rather than one of the gall-bladder; but a test meal, when possible, will usually reveal the absence of the functional and chemical signs of gastric cancer. Palpation with a sound introduced into the stomach may aid the diagnosis.

7. *Cancer of the liver* is usually secondary, and the tumor is less easily fixed with expiration than are tumors of the stomach. A satisfactory means of differentiating is, where possible, to place the hand above the tumor. Then, if the liver can be felt still higher, the tumor is probably gastric; at all events, it is not hepatic. The same is true if, with inflation of the stomach, the tumor alters its position. When the cancer of the liver is primary, the liver becomes large rapidly; jaundice is early and pronounced. The gastric signs are slight or absent, and there is no hæmatemesis nor gastrectasis. Moreover, the usual signs after a test meal are not found in the contents of the stomach.

8. *The pancreas*, either normal or pathological. When normal, it is usually found deeply situated in the median line, fixed and immovable with respiration, and disappearing upon inflation of the stomach. When it is diseased, and especially when it is the seat of a cancerous growth, one may find clayey, fatty stools, even when there is at the same time no jaundice. Jaundice may, however, be present from pressure of the growth upon the common duct. The portal vein may be pressed upon, and the resulting obstruction may give rise to ascites. There may be glycosuria, and, as a rule, the course is a rapid one. Armstrong recently reported a case in which a large pancreatic cyst simulated cancer of the stomach, even to the analysis of the gastric contents. The stools, however, had not been observed.

9. Other conditions which may simulate a gastric tumor are gumma of the left lobe of the liver, aneurism of the abdominal aorta, swollen glands about this artery, a movable kidney, and enlarged spleen, the last simulating a cancer of the fundus.

Tumors within the Stomach.—*Gastric tumors* arise either from carcinoma or sarcoma, cicatrized ulcer of the stomach or duodenum, with or without a perigastritis, thickened or spastic pylorus, fibroma, lipoma, or foreign bodies (hair ball, gastroliths, etc.).

The differential diagnosis depends upon the history, the symptom complex, and the summing up of the results of physical examination:

1. *In scarred pyloric ulcers*, stenosis and gastrectasis occur with the results common to all cases of pyloric obstruction, but the history of previous ulcer, the long duration, the presence of a smoother and more movable tumor, the character of the vomitus with proteids well digested, and chemical findings typical of benign stenosis, usually render the diagnosis clear. There are, moreover, no metastases.

2. *A hypertrophic stenosis of the pylorus* is usually acquired, but occurs, however, oftenest in early infancy. The cases late in life usually have diagnostic features common to scarred pyloric ulcers with obstruction with which, too, they have usually a causal connection. Simple hypertrophy is undoubtedly rare. Sometimes it occurs with mere hypersecretion. One relies on the history and duration, the chemical analyses, and absence of general signs of cancer. Occasionally it may be impossible to differentiate the two conditions.

3. *Spasm of the pylorus* may possibly induce gastrectasis, and if so will manifest few if any of the signs of malignant disease. The condition will be of long duration, with exacerbations and remissions, and will present no chemical signs in the gastric contents of malignant disease nor occult bleedings in the stools.

4. *Fibromata and lipomata* can only be inferred and not definitely diagnosed in the present state of our knowledge.

Regional Diagnosis of Tumors within the Stomach.—Certain differential features are useful to determine *the exact site* of the neoplasm in the stomach.

Cancer of the lesser curvature usually presents achylia from the beginning. There is less motor insufficiency until cancerous infiltration occurs. Peristalsis is absent, but one may get tonic contraction of muscle (gastric rigidity). When the neoplasm extends to the pylorus, we get pyloric rigidity with insufficiency, *i. e.*, a paradoxical condition of motor insufficiency with pyloric insufficiency, which may be tested by inflation. It is usually a late sign.

Cancer of the lesser curvature is palpable only if ptosis exists or if the tumor has attained a considerable size. The weight is often maintained for a long time, because there is less motor insufficiency. Salomon's test has special value in cancer of the lesser curvature without pyloric stenosis, although it is useless if the cancer has developed on an old ulcer, because small amounts of HCl will digest the albumin. Estimation of the ferments aids regional diagnosis sometimes. The fact that the fundus yields pepsin and rennet, while the pylorus gives merely pepsin, affords an opportunity by ferment estimates of locating the probable site. If rennet is preserved while the pepsin is relatively diminished, it implies *involvement of the pylorus*, while if both are lessened it signifies a tumor *of the fundus*.

Cancer of the Cardiac End.—Statistics of the Middlesex Hospital (1854 to 1904) record 227 cases of cancer of the stomach, of which 19 had their origin at the cardia; 13 were in males and 6 in females. Certainly the condition is uncommon, and many cases supposedly originating in the cardia have probably commenced in the œsophagus. Fagge's views expressed in his reports from Guy's Hospital on the frequency of gastric cancer suggests that "almost all cases that have been set down as examples of cancer affecting the cardia have really been instances of cancer of the end of the œsophagus extending into the adjacent parts of the stomach. . . . Indeed, on a priori grounds we should expect that a part at which the digestive tube is opening out into a large cavity should have little or no tendency to be affected with the disease in comparison with the narrow passage above it."

The type of cancer originating at the cardia is usually adenocarcinomatous. Sometimes a cardiac cancer with stenosis is accompanied by another separate and independent cancer of the pylorus, and sometimes, too, cancer of the cardia is accompanied by a secondary stenosis of the pylorus from pressure of metastatic periportal glands.

Symptoms of Cancer at the Cardia. Not infrequently these remain latent for a long time and certainly in view of their position beneath the liver and ribs they are not accessible to palpation unless they have become extensive or unless there be an accompanying gastropnoia. An interesting example under personal observation occurred in a man, aged forty-eight years, whose death occurred within four weeks of the onset of the symptoms. His earliest symptoms were those of abdominal distention, swelling of the left leg, and some weakness. Careful inquiry elicited the fact that while for three weeks he had felt general abdominal pain with some anorexia, there had been no other subjective evidence of gastric disease. At the end of three weeks (on admission to hospital) there was enormous distention from ascites, and he died two days later, sudden abdominal pain appearing; the autopsy revealed a primary cancer of the cardia, with secondary involvement of the peritoneum.

As a rule, in addition to the general signs of malignant disease, there is dysphagia and distress immediately after meals with regurgitation of food and mucus. Pain comes on at once after food, and is usually greater in this form than in cancer of the pylorus, and often there is tenderness on percussion or pressure over the xiphoid cartilage. As obstruction develops at the lower end of the œsophagus, there is at first merely some difficulty in the ingestion of solids, and their deglutition is assisted by liquids. Oppression and discomfort usually supervene. Later on, even soft foods are swallowed with difficulty, and finally only liquids can be taken into the stomach. When the obstruction is greatest, all food is ejected with retching. Vomiting is less frequent than mere regurgitation.

The passage of a tube meets with an obstruction near the cardiac orifice, although it should be remembered that in the absence of cancer, resistance is often felt from mere spasm of the cardia. The withdrawal of blood on the end of the tube is of great diagnostic value. There is usually no motor insufficiency, and lactic acid fermentation need not exist. The delay of the deglutition sounds is not a reliable sign. A tumor is not always palpable unless there be ptosis of the stomach, although with extension of the growth, the edge may sometimes be felt on deep inspiration.

Course and Duration of Gastric Cancer.—Dyspepsia, loss of flesh, pain, and vomiting, in the order mentioned, mark the progressive course of the malady. As a rule, the disease has few or no remissions, and the progress to a fatal ending is uninterrupted. Sometimes temporary improvement, both subjective and objective, occurs with perhaps marked gain in weight, color, appetite, and even strength. These ephemeral remissions often render the differential diagnosis from chronic gastritis most difficult. Sometimes there is an acute rapid course with vomiting and a fatal termination in a few weeks, with carcinosis. Schweppe, in 1890, had collected 22 cases from the literature, and since then numerous others have been observed.

The duration varies according to the nature of the neoplasm, its site and extent. Non-obstructive scirrhus cancers are the slowest to develop, while medullary cancers and those causing stenosis of the orifices proceed more rapidly. Some authorities regard the cases which occur in youth as being especially rapid, but statistics do not all bear out this view. The average duration in cancer of the stomach is about one year, although some patients live longer than three or even five years. Death occurs either from marasmus, metastases, or complications (hemorrhage, perforation, peritonitis, sepsis, collateral inflammation, *e. g.*, pneumonia, etc.).

Prognosis.—This is always extremely grave, and few cases are cured. The medical treatment is purely symptomatic, and permanent relief is only to be had from complete resection of the malignant tissue. This interference is unfortunately suitable in but a limited number of cases, although with improved methods of establishing an early diagnosis and better surgical technique, the proportion of cures show a satisfactory advance in the last five years out of all proportion to the gains in all previous years.

Treatment.—Medical.—This varies chiefly according to the situation of the growth and the functional signs. While the radical treatment is essentially surgical, there is much to be said for the medical treatment, which may be of great benefit, even though it cannot in any sense cure the condition. It is especially called for in inoperable cases, or where for some reason operation is refused, and finally in those cases where a palliative gastro-enterostomy has been performed and the physician is called upon to aid in alleviating the distressing symptoms which inevitably occur under such conditions. The cases may be divided into (1) those with obstruction at the cardia, (2) those with pyloric stenosis, and (3) those in which no orifice is involved, but the body of the stomach is the main site of the neoplasm.

The treatment is dietetic, mechanical, and medicinal.

In *cancer of the cardia*, especially with the development of obstruction, soft or liquid diet is essential, and solids should be avoided. Small amounts with frequent feedings are better than large meals, and will better prevent regurgitation, pain, and distention of the oesophagus. Mechanical treatment plays but little part, and the use of a sound for therapeutic purposes is of questionable value. There is great danger of increasing the distress by injuring the parts, although if a tube of proper size and consistence be carefully employed it sometimes gives freer passage to food for a time and relieves symptoms.

In *pyloric stenosis with gastrectasis* the diet should not be too restricted. As a rule, liquid foods which are easily assimilated are best borne. But one should be largely guided by general principles, consulting the preferences of the individual.

The meals should be small, frequent, and non-irritating. Hydrochloric acid being usually deficient, meats and other albuminous foods should be restricted. When administered they should be finely divided, and free from cartilage and other ingredients difficult of digestion. White meats are preferable, chicken, game, and pigeon, also veal and beef in small amounts, and fish. It is well after the use of proteids to give small quantities of dilute HCl (unless of course HCl be present already in the gastric secretions). Fleiner recommended the use of sauces containing HCl, *e. g.*, beef juice with warm water, to which ten drops of dilute HCl have been added. While it is true that the use of HCl is theoretically of little value, as it cannot duly replace the deficiency of the secretions, yet experience teaches that it often aids digestion and makes the ingestion and assimilation of proteids more easy. The same may be said of the use of pepsin and pancreatin, which, despite the observations of Chase and others, gives a sense of comfort to the patient after the food has been taken. Instead of HCl, Eichhorst uses phosphoric acid after meals, and if pain be present he recommends the addition of small doses of codeine.

It is a delusion to believe that the liberal use of albuminous foods prevents decomposition of the albumin of the body. The more a cancerous patient gets, the more he destroys.

The less coarse vegetables, preferably mashed, may be used in relatively large amounts, avoiding those with shells, skins, fibers, etc., as also those which induce much fermentation. These should be seasoned to aid the appetite. Those most to be preferred are spinach, cauliflower, mashed potatoes, asparagus tops, well-cooked turnips, and carrots. Stale bread may be allowed, as also gruels, vegetable soups, and thin, farinaceous preparations like rice, macaroni, barley, tapioca, sago, and oatmeal. Stewed fruits are also well taken. Milk with coffee, cocoa, cereals, nutrose, eucasin, tropon, plasmon, etc., may be employed, as also buttermilk and koumyss. Infant foods are often taken with relish, and should be tried. Butter is the best borne of all the fats.

As beverages, one may use any of the ordinary drinks in use, but the amounts at each feeding should be limited on account of the motor insufficiency. Light wines, brandy, and whisky may be taken in small amounts and diluted. Thirst is often a troublesome condition, and when it remains unassuaged by the moderate use of fluids, saline enemata may be employed with benefit, one-half to one pint being injected night and morning.

The mechanical treatment consists of the judicious use of lavage with plain water. As a rule, this alleviates the distressing local symptoms, and may help to increase the appetite and stimulate the secretions. The washings should be thorough at each occasion and unless gastrectasis be extreme, one lavage daily will suffice. As a rule, the most suitable time is before the last small evening meal or in the early morning. Cohnheim introduces two or three ounces of warm oil through the tube after the completion of the lavage, thereby giving still greater relief to the local distress.

In *cancer of the body of the stomach*, where motor insufficiency is but little evident, the main treatment is dietetic. Food administered as above described in pyloric stenosis, with small frequent meals of easily digested food, given according to the secretory conditions, will often improve the patient's general condition, relieve the symptoms, and even add to the general nutrition and weight. In these cases mechanical treatment is less indicated.

Nutrient enemata are to be employed from time to time when food is taken in insufficient amounts or when vomiting is persistent.

Medicinal Treatment.—No medicine is of any great value. Orexin in doses of 5 grains (gm. 0.3) may help the appetite, as also may an infusion of condurango bark. One finds that alkaline mineral waters before meals sometimes stimulate the secretions, while HCl after meals helps proteid digestion. For the *vomiting*, especially of blood, rest to the stomach is essential. Nutrient enemata may be given, and then careful feeding by mouth may be begun with small quantities of fluid. For the ordinary vomiting, lavage is the best form of treatment.

Diarrhœa should be treated by a proper diet when possible, and by the use of lavage to remove irritating substances, which are the probable cause of the condition. For the *constipation*, aloes, rhubarb, and cascara are the most effective remedies. Enemata should be given when the condition is aggravated, but these should not be continued for too long a time. Authorities differ as to the use of saline purgatives, and doubtless in individual cases their use will have different effects.

For the *pain*, wet compresses upon the stomach, lavage, and the use of spirits of chloroform may be of value, and it is only in the extreme cases that morphine should be employed.

Surgical Treatment.—Cancer of the stomach being recognized as a disease which in the present state of our knowledge is only remediable by early removal, the curative treatment is purely a surgical one. When we realize that 9000 deaths from cancer occurred in the United States in 1900, and 5000 die yearly from that disease in England and Wales, the importance of proper treatment becomes more evident. Few cases are seen sufficiently early. Boas mentions 243 patients with cancer of the stomach of whom only 60 were seen in the first three months from the onset of the symptoms, and of these only 3 came to resection.

Billroth first successfully operated in 1881, although as far back as 1810, Merrem (quoted by Robson) showed the possibility of successful removal of the pylorus in dogs. Statistics on operation for gastric cancer are for the most part unsatisfactory, especially those of earlier years, and "successful" operations often imply nothing more than immediate recovery after the operation, quite apart from the possibility of subsequent recurrence or death.

Operations are undertaken in cancer of the stomach under two main conditions:

1. *Exploratory for diagnosis*, and not merely to confirm the diagnosis. When a tumor is present and the gastric analysis gives every indication of cancer, it is but idle curiosity to explore, unless it is known beforehand that some chance of giving relief exists. The object of exploration is to *make* the diagnosis when great suspicion and doubt exist as to the possibility of cancer. If in such cases a reasonable suspicion exists, the earlier discovery which an operation thus permits, gives to the patient the better chance of radical surgical cure, and as such, exploration is not only justifiable but advisable in the extreme. Delay under these conditions, to await further evidence, implies unnecessary waste of time and a further opportunity for extension of the growth.

At other times it may seem uncertain (even when a tumor is palpable) whether extirpation is advisable and possible, and under these circumstances, too, exploration may be considered justifiable. Lastly, inasmuch as all gastric cancers present features of uncertainty until the abdomen is opened, it may be said that whenever operation is decided upon, the primary incisions are purely exploratory and the later course of procedure defined only after exploration has been made.

2. *Operation for treatment*, either curative or palliative. Suitable cases require that the motor power be sufficiently maintained to enable the food to pass on into the intestines, where the main digestive functions will thereafter be performed. Should any portion of the stomach be retained in the resection of the tumor, it may be so diseased as a secondary result of the cancerous growth that its function has quite ceased and the secretions are either absent or useless. For this reason the preservation of a good motor power is an essential to a successful operation, for even although secretions are sometimes really restored, this does not apply invariably, and, in fact, is the exception, not the rule.

The cases which promise successful results are as follows:

1. Those cases in which the neoplasm is well localized and movable. It is true that some very movable tumors are adherent by their posterior surface, and in these cases one should be guided chiefly by the character and extent of the adhesions, those which are not extensive and uninfiltated

being most satisfactory to deal with. Hence, Ewald's contention that one must explore before deciding on further operative interference.

2. Well-localized tumors which are free from much lymphatic involvement; extension to neighboring organs contra-indicates operation (unless it be in the transverse mesocolon, in which case the area involved is often easily removed by a wedge-shaped incision). Lymphatic involvement is not always easy to estimate, and glands which are enlarged in the vicinity of cancerous tumors are often merely infected and inflamed, not truly cancerous. This is especially true with ulcerating cancers.

In the stomach itself the course of the lymphatics has an important bearing on the prognosis and treatment. As has already been said, the studies of Cuneo have shown that the lymphatics along the lesser curvature run for the most part from the pylorus *toward* the cardia, while those along the greater curvature and fundus lead toward the pylorus. Thus in operations for gastric cancer, the extent of removal depends largely on the site of the growth. Pyloric cancers do not tend to spread to the fundus and greater curvature, and these parts, therefore, do not need to be included in the resection. On the other hand, the lesser curvature should be removed, at all events as far as the gastric artery (Mikulicz's point), and along the greater curvature as far as Hartmann's point, which delimits the lymphatic extension along that line (Hartmann-Mikulicz's line of section). Toward the duodenal end, the resection should be made one inch clear of the pylorus, this being the extreme limit of secondary involvement in nearly all cases.

It should be remembered that even in cancers of long duration the neighboring glands are sometimes left free, just as occurs sometimes in rodent ulcer. Debove and Le Riche have recorded cases of this kind in which, after ten years' duration of symptoms, a cancerous tumor with adhesions was found, but the glands were unaffected and there was no generalization. It may be questioned whether such a condition was primarily cancerous, or whether the original lesion may not have been of a simple ulcerative character.

Special Considerations.—*Infiltrating adhesions* are a contra-indication and a dangerous complication in operations. It has been estimated that 73 per cent. of such cases die, and the remaining 27 per cent. recover only because the adhesions were very slight indeed, and probably non-infiltrating.

The Size of the Tumor.—The fact that a tumor is externally palpable is no longer regarded as a contra-indication to surgical treatment. In fact, the size of the tumor is less important than is the presence of adhesions. Many large tumors may be capable of successful removal because they are free from adhesions and metastases, while many primary tumors which have remained very small may have involved neighboring glands and organs. Metastases in the liver, for example, may be large and numerous even when the primary local gastric neoplasm is small, movable, and non-adherent.

Complications form another important contra-indication, *e. g.*, arteriosclerosis, fatty heart, and tuberculosis.

Statistics from the various surgical clinics are published from time to time, showing the average duration of life after operations of various kinds. These, of course, have no value as regards the selection of operation. To know, for example, that after a gastro-enterostomy, 33 per cent. died is of little value. Such an operation is merely palliative and the cancerous growth proceeds. Immediate results depend largely on methods of technique and the condition of the patient at the time of operation, both of which

factors determine the success of operation and the ultimate duration of life. The only operations which are curative are those which remove the entire diseased parts. In other words, gastrectomy, partial or complete, is the only procedure which assures success, while other operations are merely palliative.

Methods.—*Gastrectomy* was first successfully performed by Billroth in 1881, when a partial operation was done, while complete removal of the stomach was first attempted by Connor, of Cincinnati, in 1883, who, although unsuccessful, paved the way for the later good results of Schlatter, of Zurich, in 1897, who performed a complete gastrectomy with recovery. The œsophagus and jejunum were united and the duodenal opening closed. Since then gastric operations have become matters of every-day experience and the European clinics especially give large series of cases with statistical results (Kroenlein, Mikulicz, Kocher, Czerny, Robson, and Moynihan).

H. J. Paterson collected some statistics on gastrectomy up to 1905; 27 cases had been operated upon for complete removal; 10 of these patients were alive and well at varying periods up to eight years after operation (eight, seven, five, four and three-quarters, four, three and one-half, two, one and three-quarters years, respectively, and two others for a somewhat shorter time). Ten others had died, of whom three had lived more than one year (*i. e.*, mortality 36 per cent.). Twenty other cases were recorded, in which subtotal gastrectomy (*i. e.*, leaving a portion of the fundus) was performed, and of these only 6 had died (mortality 30 per cent.), while all the others had lived, and 9 of them had survived the operation for from two to eleven years. Paterson's statistics include, further, 55 cases of partial gastrectomy (*i. e.*, removal of the pylorus and the usually lesser curvature). Of these, 14 per cent. have been cured, having survived the operation without recurrence (1 fourteen years, 1 seven and a half years, 2 six years, 1 five years, 2 four years later, all well; 1 four and one-half years, but with suspicion of recurrence).

Partial gastrectomies result in improved motor power; secretion is not altered, as a rule, although sometimes HCl returns and lactic acid disappears when stagnation ceases. Modern surgery advocates partial gastrectomy as the best of all operations, as yielding less mortality and because it results in better health, comfort, and appetite, and in general well-being, with the best chances of complete recovery. Mayo quoted MacDonald as having found 43 undoubted cures from operation. Kocher, in May, 1907, reported 95 cases, of which 13 were well, and 4 others, although cured of cancer, had died later on from other diseases, *i. e.*, a total of 17 (19.3 per cent.) cured. Le Riche has recently tabulated cases, of which 89 cancers with gastrectomies were cured for a period varying from five to sixteen years. Of these, 1 had survived sixteen years, 34 from five to ten years, and 5 at least ten years. These cases include all kinds of cancers anatomically, mostly from the pyloric region, but one had been at the cardia. In a total of 1366 collected gastrectomies for gastric cancer, the mortality was 25 per cent. Peterson and Colmers recorded 30 cases with 20 per cent. of cures (Lisbon Congress), adding that all these patients were then in good condition, performing ordinary daily work with no gastric complaints, with an easy digestion, and that even the distaste for fats and meats had disappeared. The secretions, however, were feeble. In all there was deficient HCl, some still had lactic acid, but the motor power was excellent. Undoubtedly, such statistics are

more than favorable, and, in view of general experience, extremely optimistic. It should be added, however, that much probably depends on the virulence of the cancerous growth and the susceptibility of the individual.

Remote Results.—Evidently the mechanical, physiological, and chemical functions of the stomach can be taken up by the parts left behind. Sometimes the œsophagus or small bowel dilates to form a kind of reservoir. Digestion is carried on well by the intestinal juices and pancreatic and hepatic secretions, and absorption takes place in an apparently satisfactory manner, and thus perfect health is maintained. Hayem has observed “hypopepsia” after years in patients in whom cures have resulted following operations for gastric cancer.

Palliative surgical treatment is employed in some inoperable cases where the strength of the patients permits surgical interference if the indications therefore justify the undertaking. Cure is not aimed at, but merely a relief of certain symptoms and fulfilment of certain requirements for improvement of local and general conditions.

In one class of cases, *e. g.*, with pyloric stenosis and motor insufficiency, a *gastro-enterostomy* is advisable to relieve stagnation of food, vomiting, etc., by improving the drainage of the stomach contents. At the same time it sometimes removes the pain due to increased peristalsis and irritating secretions, as well as the danger from the toxæmia of absorption. In another set of cases, *e. g.*, with cardiac stenosis, food cannot enter the stomach in sufficient amount to maintain nutrition and life, and a *gastrostomy* is done to permit introduction of food into the stomach by means of an artificial gastric fistula.

Gastro-enterostomy, first performed by Wölfler on the suggestion of Nicodoni, in 1881, is now recognized as a safe operation when done in accord with modern methods and proper technique, and in cases where the general and local conditions are suitable for such an undertaking. In Chlumsky's statistics of 550 cases (1881 to 1896), 42 per cent. died, whereas Mikulicz, in more recent reports, had a mortality of only 28 per cent., and still more recent are the statistics of Robson, with a mortality of only 1.5 per cent. in 200 cases of gastro-enterostomy for cases of ulcer.

The indications for gastro-enterostomy are, briefly, pyloric stenosis from a cancer which cannot be removed, either because of its extent or owing to the involvement of too many neighboring glands, hour-glass or other deformity involving stagnation of food and its complications, hemorrhage which is persistent, and uncontrollable vomiting, whether from retention or from general causes. It is further indicated in some cases as a temporary measure prior to the more serious operation of gastrectomy, where patients are too weak to undergo the latter operation immediately.

In any doubtful cases, with adhesions to the neighboring organs or pressure of inflammatory tissues about the pylorus, a gastro-enterostomy may not only relieve, but cure. In a patient whose condition was recently reported by my colleague Dr. Garrow, a large indurated and nodular mass was found at operation involving the pylorus. It had been tucked up underneath the liver, and thus was not palpable; a second mass extended along the lesser curvature, while the glands in the lesser omentum were enlarged, although not indurated, as was the mass in the pyloric region. The condition was regarded as undoubtedly of a cancerous nature, and a palliative gastro-enterostomy was performed. One year later the patient was seen again in

excellent health, suggesting the probability of some lesion other than a malignant one. Operation should be delayed where there is little gastric disturbance and where there is great weakness. Cases, too, with small stomachs and infiltrating cancers should be let alone.

The beneficial results of gastro-enterostomy are often marked. Patients may gain in weight, the appetite may increase, vomiting and pain diminish, and general improvement follow for some months. Comparative comfort may thus be attained. In other cases, however, results are discouraging and the patients decline rapidly in health, and the rapidity of growth in the neoplasm is marked, even closing the artificial opening.

Certain complications arise after gastro-enterostomy, which for the most part may be obviated by proper technique and the selection of suitable cases. Of these, the most important is the establishment of a vicious circle, with regurgitant vomiting, a sequel that is only the result of faulty technique. Other complications are likewise for surgical rather than medical consideration, *e. g.*, contraction of the newly formed opening, kinking of the anastomotic opening, ulceration, hemorrhage, adhesions, and hernia. The after-effects are of little account. The chemicophysiological changes after gastro-enterostomy are insignificant, and absorption is but little impaired.

Gastrostomy is indicated where food cannot be properly taken into the stomach, either through cancer of the œsophagus or of the cardiac end of the stomach. The operation should be done early to obviate the great weakness resulting from defective nutrition, and thus the ordinary dangers from a comparatively simple operation may be avoided.

Jejunostomy.—Maydl's operation. The results are not very satisfactory. Out of 22 cases, there were 4 deaths, 13 lived longer than four weeks, and of these, 7 died in three months, and 1 in a year.

ACUTE GASTRITIS.

Definition.—An acute inflammation of the gastric mucosa, with varying degrees of local and constitutional disturbance.

Classification.—Various types exist dependent upon the different etiological factors. *Clinically* there are three main types: (1) Acute simple gastritis (primary and secondary). (2) Phlegmonous gastritis. (3) Toxic gastritis (mild and severe). *Anatomically* one may add two other forms: (4) Mycotic gastritis. (5) Diphtheritic gastritis.

Acute Simple Gastritis.—**Definition.**—This is an acute catarrhal inflammation of the gastric mucosa, frequently accompanied by excessive secretion of mucus, desquamation of epithelium, and a resulting disturbance of digestion.

The condition is more often wrongly than correctly diagnosed, and in numerous instances a functional disturbance with slight temporary atony, or a neurosis, is misnamed gastritis. The term gastritis is thus often misapplied, and an examination of the mucous membrane in many of the suspected cases would doubtless reveal but little disturbance of inflammatory origin. The name should be restricted to those patients in whom definite evidence of gastric inflammation exists, either in the etiological history, or in the symptoms and signs, and should not necessarily be applied to temporary disturbances of function resulting from slight indiscretions in diet. Certainly in a very large proportion of cases it is impossible to decide

clinically whether or not a true inflammation exists, for after all there are very few pathognomonic symptoms and signs which prove conclusively the existence of a mild simple gastritis. Sufficient evidence of this is presented in the autopsy room, where the very common occurrence of acute inflammation of the gastric mucosa is found without a previous history of gastric symptoms, and vice versa, where much disturbance of digestion may have occurred and when even much mucus has been secreted, there may be but little pathological evidence of inflammation.

Etiology.—The condition is either primary or secondary.

Primary Acute Simple Gastritis.—Predisposing and direct causes exist. The former are numerous and include faulty hygiene, the hot summer season, malnutrition, anæmia and other circulatory disorders, constitutional and metabolic disturbances, and infectious diseases; tuberculous patients are particularly susceptible; and a previous chronic gastritis predisposes to acute attacks. Individual susceptibility is an important factor, and perhaps heredity plays a part. In one instance under the writer's notice poultry of any kind invariably induced an attack, no matter how modified by cooking. Age too has much to do with susceptibility, infants being peculiarly liable.

The *direct causes* are mechanical, thermal, chemical (toxic), or parasitic (vegetable or animal).

1. **Mechanical causes** are mainly those associated with errors in diet, *e. g.*, irritating food or drink, too bulky or coarse food, or excess after prolonged fasting. Food when taken in too large an amount may remain undigested, and induce fermentation or putrefaction, so, too, the shells of some vegetables, the stones and rind of fruit, foreign bodies, hair, etc.

2. **Thermal Causes.**—Food and drink if at extremes of temperature may induce gastric catarrh, so also the external application of heat and cold; extensive burns on the surface of the body for example, are frequently followed by catarrhal gastritis, the cause remaining unexplained. Whether or not "cold" is a cause per se is doubtful.

3. **Chemical causes** are perhaps the most frequent. Alcohol is the commonest factor in producing acute gastritis and leads to the disease in its most typical form. Those unaccustomed to alcohol are the most liable, and the inflammation arises either from irritation from the amount taken, or in the case of some wines because of the acid produced by acetic fermentation. In children unripe fruit is a common cause.

Any chemically strange substance if toxic, in strong enough concentration and of sufficiently irritating character, will induce a gastritis, *e. g.*, alcohol, acids, alkalis, salts of the heavy metals, calcium chloride, and certain drugs, *e. g.*, salicylates, the bromides, iodides, arsenic, mercury, phosphorus, copaiba, etc.

Decomposed pus, as in gangrene of the lung when the irritating sputum is swallowed, and, indeed, any decomposed albumin may directly or indirectly induce the disease.

Foods if tainted or decomposed ("crapulous gastritis") bring about an inflammation directly through the action of fermentative or putrefactive agents; so also does stale beer.

Toxalbumins form another group of agents. They are secreted in the stomach from a variety of sources, *e. g.*, in Asiatic cholera. Such agents may be denominated as autotoxic causes, and are present in numerous diseases (uræmia, diabetes, gout, cholæmia, and also in burns).

4. *Parasitic Causes.*—*Vegetable Parasites (Bacteria).*—The invasion of bacteria is one of the most common causes of acute gastritis—hence its frequent presence in the various acute infections of all kinds. The colon bacillus is one of the most frequently found organisms, and in cases where it occurs in the course of acute infections is often regarded as the cause of the secondary gastritis. In all such instances the origin of the gastritis may be largely chemical, and due to the products of the bacteria. An epidemic form of gastritis has received attention from various writers who regard this ill-understood condition as originating in some form of microbic infection. This “gastritis infectiosa” is regarded by some as a special disease.

Animal parasites are commonly associated with a true gastritis, although it is by no means a necessary feature of parasitic invasion of the stomach. The ascarides, the tænia, and even the oxyurides may induce an accompanying simple catarrh of the gastric mucosa, as may also the diptera, flies, brachycera, and larvæ of various kinds. Gastritis is not uncommon in malaria.

Secondary acute gastritis develops in the course of many infectious diseases, and may indeed be the main initial symptom to arouse suspicion of a grave infection. This is especially the case with children. In various pulmonary diseases when degenerated or decomposed tissues and exudates are introduced incidentally into the stomach an acute catarrh results. Ellis¹ records a hemorrhagic gastritis due to a swallowed laryngeal pseudomembrane, which also obstructed the pylorus. As an accompaniment of various intestinal diseases, acute renal and cardiac disease, etc., acute gastritis is not uncommon.

Pathology.—The size and shape of the stomach are not usually altered. There is a more or less diffuse redness and swelling of the mucosa, with congestion or patchy hemorrhages and an excessive quantity of mucus. The pyloric region is most frequently involved.

Microscopically, muciparous, cylindrical epithelium covers the free surface of the mucous membrane as well as the relatively deep stomach cells. The glandular epithelium itself shows granular degeneration, with more or less fatty change and atrophy, and the lumen contains debris, for the cells themselves have desquamated. Hyperæmia occurs either diffusely or in patches, and there are more or less minute hemorrhages. Leukocytes have escaped from the surrounding dilated capillaries, and a certain amount of small-celled infiltration is evident in the interstitial tissue. The solitary lymph follicles may be swollen, and sometimes burst on to the surface leaving minute ulcers.

The resulting changes affect the secretion of mucus and gastric juice, and alter the general functions. Hence disturbances of the sensory, secretory, absorptive, and motor gastric functions occur.

Symptoms.—The symptoms depend much upon the cause. Usually within a few hours, or at most a couple of days after action of the causative agent, there is gastric discomfort, fulness and sense of pressure or pain, with pyrosis, nausea, and vomiting. Often an initial rigor or chilliness ushers in the disease.

¹ *Transactions of the Pathological Society of Philadelphia*, 1903, new series, vol. vi, p. 197.

The patient complains of a curious taste, sometimes bitter; there is also salivation. Thirst is a prominent feature. Nausea usually precedes the vomiting and occurs with food, or at the mere thought of it; vomiting itself occurs early and gives relief. The vomitus contains undigested food, mucus, bile, and sometimes streaks of blood. It is bitter in taste, and, as a rule, of a foul odor. Cardialgia, pyrosis, and waterbrash add to the distress. The teeth are set on edge and the burning acid fluid irritates the pharynx and œsophagus. There is hiccough, and eructations of gas, either bitter or tasteless, are common. Pain is almost always present, slight or severe, and sudden or gradual and progressive. With all these subjective sensations are numerous nervous phenomena, headache, frontal, occipital or temporal, often described as a hammering headache, dizziness (vertigo), dulness and depression of spirits.

Signs.—The patient shows more or less prostration, and, if the condition be at all severe, the face is pale, the extremities are cold, and he breaks out in a cold and clammy perspiration. The pulse is at first increased, later lessened in rapidity. Fever, if present, appears early, and is usually slight, although sometimes, when there is an initial chill, it may be high and accompanied by herpes; often, indeed, there is no fever throughout, but when, on the other hand, fever persists for days, one should examine carefully for some other factor to which gastritis may be merely secondary. In the so-called epidemic or infectious gastritis the fever remains high for days—sometimes even for several weeks. The condition differs otherwise but little from ordinary simple gastritis, although an enteritis is a much more frequent complication and the general symptoms are more severe.

The tongue is coated, usually dry, indented, and the edges come in contact with the teeth. The breath is foul and salivation is marked. Herpes labialis is very often present. The epigastrium shows distention and tenderness. The vomitus contains HCl with lactic and fatty acids, and mucus, and there is motor insufficiency of a mild degree. The test meal likewise shows evidence of stagnation and perhaps subacidity, while mucus is excessive. After a test breakfast the bread is almost unchanged, much mucus is present, and HCl is absent or much diminished. There is usually constipation or later on diarrhœa, and involvement of the intestines is an important and often troublesome complication. Jaundice may be present if the common duct is obstructed by a plug of mucus. The urine is scanty, high colored, has a high specific gravity, and shows abundance of urates.

Diagnosis.—The etiology is an important factor, while the acute onset of the symptoms and the reaction to treatment suffice to make the condition clear. The absence of a known and plausible cause should suggest caution in a diagnosis which may subsequently require modification.

The *differential diagnosis* deals mainly with gastric neuroses, neuralgia, and hyperacidity, hysteria and ulcer. Further, biliary colic, catarrhal jaundice, appendicitis, incarcerated hernia, ileus, and the onset of some infectious disease, especially typhoid fever, scarlatina, smallpox, and pneumonia. The gastric crises of tabes dorsalis and the onset of meningitis in children, and lastly pregnancy should not be forgotten in considering the differential diagnosis, for many awkward mistakes are thereby avoided.

The appropriate adjustment of the history and symptoms to the physical findings will usually in time make the diagnosis simple. *Exclusion of*

the important causes of a secondary gastritis is the essential feature in the diagnosis.

Prognosis.—The prognosis depends on whether the condition be primary or secondary, and the gravity of the associated underlying cause. As a rule, primary acute simple gastritis is quickly cured by appropriate treatment. The more frequent the attacks, however, the greater the liability to recurrence.

The *course and duration* depend very largely on the cause and severity. The symptoms may last merely for twenty-four hours, or in severe cases for several weeks. Sometimes vomiting relieves at once, but such cases are in most instances merely acute dyspepsia, *i. e.*, acute disturbances of function. When the intestines are involved the disease is aggravated and its duration lengthened. Fatal cases are those in which the gastritis is secondary to some other disease, usually some severe infection or intoxication.

Treatment.—*Prophylaxis.*—In children the cause is more frequently dietetic than in the adult, and in them proper feeding is an important prophylactic measure. The overloading of children's stomachs with unsuitable food, especially uncooked and unripe fruits, is a frequent cause of gastritis.

The Direct Treatment.—Two considerations are of special importance: (1) To remove the cause. (2) To protect the stomach and lessen the inflammation.

1. Nature often provides for the removal of the cause by inducing vomiting, and subsequently anorexia, or else emesis may be produced by some simple measures. The remnants often irritate and keep up the symptoms, and are liable to add intestinal disturbances, so that frequently it is well to wash out the stomach; this is especially useful with children because thorough and rapid in its effects.

Where vomiting is not rapidly induced one may use emetics with care, apomorphine hypodermically, or a mixture of tartar emetic, gr. $\frac{1}{4}$ (gm. 0.016), and pulv. ipecac, gr. 5 (gm. 0.3), every quarter of an hour until it acts. The latter drug is especially useful for children in the form of vin. ipecac. in $\mathfrak{z}\text{j}$ doses. Other medicines are rarely required.

2. For twenty-four hours the stomach should be given as complete a rest as possible. A little chopped ice relieves the thirst and a suitable mouth wash should be in frequent use. Whatever else is given should be in small quantities and only in fluid form, for example a little weak, unsweetened tea, perhaps with brandy; effervescent water or soda lemonade; peppermint tea; later milk and lime-water (or soda-water) may be taken, other food being begun only when the desire for food returns. Pancreatized milk is also useful. Broths, gruel, and a little stale bread may be added. Solid food is given later on if no diarrhoea be present, the transition period being devoted to light puddings, rice, etc.; then lean fish, and later on stewed chicken and fillet.

Light vegetables should be allowed only after some days of convalescence when the stools are quite normal, for relapses are common and easily induced by too early a return to a mixed diet. Rectal feeding is rarely needed, but is advisable for those in advanced years.

Medicinal.—The early administration of muriatic acid is beneficial, sometimes, as Riegel advises, in 8 to 10 drop doses of the dilute acid in a wineglass of water, taken in sips before meals, or else as Eichhorst pre-

fers, given after food. Narcotics are not usually required, and may do harm by stopping peristalsis and thus prolonging the symptoms.

Mechanical Measures.—A Priessnitz compress and an early warm bath are sometimes of use.

Special Symptoms.—For the constipation, drastic purgatives are to be condemned; however, small doses of calomel and soda are useful, especially for infants, or castor oil and laudanum. "Fluid magnesia" is preferable in mild cases because less drastic. For the diarrhœa, lime-water in doses of a few ounces every two hours, or bismuth and chalk in powder form may be given. For the nausea and rest to the stomach, cocaine in doses of gr. $\frac{1}{4}$ or creosote in 1 minim doses may benefit. Vomiting is best treated with rest, and the administration of dilute hydrocyanic acid in doses of 5 minims with bismuth and soda and spirits of chloroform, although, as a rule, a sufficiently prolonged abstinence will in itself suffice.

Phlegmonous Gastritis.—**Synonyms.**—Interstitial purulent gastritis; inflammation of the submucosa (Rokitansky); gastritis submucosa (Dittrich, Klebs, and others); phlegmon ventriculi; linitis plastica et suppurativa (Brinton); linitis phlegmoneuse.

Definition.—This is an acute suppurative inflammation of the gastric submucosa, sometimes primary, but usually secondary as a metastatic or other infection. It may be diffuse or circumscribed. Leith,¹ in 1896, had found 55 instances, and in 1904 Robson and Moynihan² collected 85 cases. Since then others have been described, and more than 100 cases are now on record. Varandæus, in 1620, first described the circumscribed form in his *Tractatus de Morbis Ventriculi*. Later, Borel, in 1656, and Sand, in 1701, added to the literature. The first diffuse case was recorded by Andral³ and Cruveilhier in 1839.

Etiology.—The condition is rare, especially in its primary forms, and but few cases are on record. The primary cases are doubtless all microbic in origin, although alcohol is often regarded as a predisposing factor. Such were the cases described by Welch and E. Janeway and Kinnicutt.⁴ Marasmus, faulty diet and ptomaines, corrosive poisons, *e. g.*, oil of turpentine (Kelynack), oxalic acid and trauma of various kinds, all play a part. In one instance, described by Klieneberger,⁵ the ingestion of 3 gm. of potassium iodide was followed by a marked toxic phlegmonous gastritis, no previous symptom of gastric disease having existed up to that time. Evidently the iodide had caused an intense inflammation of the mucosa and favored the invasion of pyogenic organisms. A fair proportion of cases follow gastroenterostomy and other operations on the stomach. Trauma is regarded as an occasional antecedent (Hopkins). The commoner sources of origin are some general infection, *e. g.*, scarlatina, smallpox, typhoid, etc.; pyæmia; an ulcerating cancer or infected ulcer or some defect in the mucosa admitting virulent bacteria. In a number of the cases hemorrhagic erosions suggested the site of invasion. In the opinion of some authors the infection may occur through the mucous membrane even without the presence of a defect (Konstantinovitch⁶). More rarely pus swallowed from a suppurative stomatitis has caused the disease.

¹ *Edinburgh Hospital Reports*, 1896, vol. iv.

² *Diseases of the Stomach*, second edition, 1904. ³ *Clinique Méd.*, 1839, tome ii.

⁴ *Transactions of the Association of American Physicians*, 1900, p. 127.

⁵ *Munch. med. Woch.*, August, 1903.

⁶ *Centrblt. f. Stoffwechsel u. Verdauungskrank.*, 1903, iv.

Streptococci have been the commonest microorganisms present, and the colon bacillus frequently co-exists. Many other common bacteria have been found and sometimes yeast. But, as Cecil¹ and others have pointed out, the bacteriological examination is not always satisfactory, for coccus forms may die off early, leaving the field to other organisms not directly associated with the true cause.

Age.—The youngest patient afflicted was ten years of age (Hun), the oldest eighty-five. The diffuse lesions nearly all occurred between twenty and sixty years.

Sex.—The proportion is about four males to one female.

Morbid Anatomy.—Of the 96 cases collected, 76 were probably of the diffuse variety and 12 were definitely circumscribed. The remaining cases recovered or did not come to autopsy. The disease commences in the submucosa, and unless a local ulceration (benign or malignant) exists, or some generalized infection, it is not always easy to determine the origin. Some authorities believe that in the majority of so-called primary cases an invisible or undetected lesion of the mucosa existed through which the invading microorganisms gained an entrance. Gläser,² Mintz,³ Merkel,⁴ and others have recorded cases originating in chronic ulcer and in cancer. In one of Gläser's patients an hour-glass contraction of the stomach had occurred from cicatrization of part of the ulcer prior to the secondary phlegmon. The submucosa, especially at the pylorus, is thickened, infiltrated with pus or sero-pus, sometimes almost jelly-like or œdematous in appearance. The underlying muscle is involved to a greater or less extent; indeed, it may be entirely destroyed. Sometimes merely a channel or cavity remains, containing little or no pus whatsoever. The mucosa is usually more or less affected and signs of infiltration are seen between the glandular loops. Rarely no anatomical lesion of the mucosa is visible, or, on the other hand, it may be so changed from the affection as to resemble a mass of broken-down colloid tissue.

The mucosa itself is also thickened, hyperæmic, and ecchymotic in patches. The epithelium is here and there degenerated, and erosions and ulcers are frequent. Sometimes the sinuses leading from the submucosa are so numerous as to give to the lining membrane a coarse or fine sieve-like appearance, as in a case described by Polak. The result may be that more or less of the mucosa itself is lifted away from the underlying submucous layer. The peritoneal surface shows at times marked swelling of the lymphatics. Perigastritis always accompanies the malady and adhesions occur in various ways and situations about the viscus. Purulent peritonitis is not an infrequent termination, or infiltration and dissection may occur along the submucosa of the duodenum or up into the œsophagus (Chvostek⁵). In some instances the diaphragm has been invaded, resulting in a purulent pleurisy or pericarditis. Thrombi are often found in the neighboring veins and the causative microorganism is not infrequently detected in the vessels.

Splenic tumor, acute nephritis, necrosis of the colon, mediastinitis, cholecystitis, etc., may result from the primary gastric affection, or as a part of a general septic state. The circumscribed phlegmonous gastritis forms an

¹ *Johns Hopkins Hospital Bulletin*, 1907, p. 356.

² *Bert. klin. Woch.*, 1883, p. 790.

³ *Deutsch. Arch. f. klin. Med.*, 1892, vol. xlix, p. 487.

⁴ *Centribl. f. inn. Med.*, 1905, 26, p. 257.

⁵ *Wien. Med. Presse*, 1877,

abscess of varying size in the submucosa, or there may be numerous smaller collections. Perforation may occur into the stomach cavity, externally into the peritoneum or into neighboring viscera.

Symptoms.—*The Diffuse Variety.*—The onset is nearly always sudden, although with no characteristic symptoms. A rigor may usher in the affection, followed by a rise of temperature to 104° or 105° F. There is general malaise, and severe prostration appears very early. Very soon the gastric symptoms become marked. Not infrequently two stages are evident, at first the signs of gastritis and later those of peritoneal inflammation. It must be remembered, however, that some cases show no gastric symptoms whatever, and the condition is found only after death.

Nausea and vomiting appear early, are of long duration, and rarely absent. Toward the end the vomiting usually diminishes; it may be feculent and foul in odor. The vomitus rarely contains macroscopic pus (as in the cases described by Glax, Deininger¹ and others), although one may find many leukocytes on microscopic examination of the gastric contents. The suppuration, if confined to the submucosa, as it usually is, does not set free the pus into the stomach unless the mucosa be perforated. Mucus and bile are the chief ingredients of the vomitus.

Pain in the epigastrium is a prominent feature and is rarely absent. While scarcely increased by pressure (Leube), it gets progressively worse and often becomes intense. Soon the pain extends over the whole gastric area, resembling that of local or general peritonitis. Tenderness is usually present, and sometimes one may feel fluctuation if a large abscess exist. Meteorism, which is often marked, accompanies the other signs of general or local peritonitis.

The fever is irregular, usually persistent, and has a wide range, often attaining high levels, as in other septic conditions. In Testi's case, however, there was no elevation of temperature. Repeated rigors are sometimes present. The pulse is, as one would anticipate, rapid, small, and often irregular in volume and rhythm. Diarrhœa or constipation, more often the latter, may accompany the other symptoms, and thirst is often marked. Collapse, which has been imminent from the onset, becomes progressive; delirium and coma usually usher in the final stage, with or without the signs of general peritonitis.

The course is progressively downward from the outset. The duration is rarely longer than two weeks, more commonly ending before the symptoms have lasted one week. The diffuse cases are more rapid than those which are circumscribed.

The *circumscribed variety* presents a similar type of symptoms, although at first less severe. Sometimes pain is quite absent, as in a case recorded by Callow in which the patient vomited pus, and later seven pints of purulent fluid were found in the peritoneal cavity. A distinct tumor is often felt. The abscess may burst into the peritoneal cavity and cause death, or rupture may occur through the mucosa into the stomach and pus and blood are vomited. Some of these cases recover and pathological specimens testify to this form of healing.

Diagnosis.—This has rarely been made. The signs are those of acute peritonitis (local or general). Leube has pointed out that the vomiting

¹ *Deutsch. Archiv. f. klin. Med.*, 1879, Bd. xxiii, p. 624.

of pus need not be pathognomonic and may arise from a purulent gastritis alone. The probable diagnosis is suggested when in the course of a pyæmia or other severe general infection the symptoms arise as above described, although this form of metastatic infection is comparatively rare. The circumscribed form is perhaps more readily diagnosed, especially when a tumor, which has already been felt, disappears after the vomiting of pus; yet even this may lead to false conclusions when an abscess arising outside of the stomach perforates into that viscus. Leukocytosis is probably present in most of the cases (Lengeman).

In the differential diagnosis one must consider chiefly perigastritis following ulcer, peritonitis, acute pancreatitis, suppurative cholecystitis, abscess of the liver, and corrosive poisoning.

Prognosis.—About 95 per cent. of the recorded cases have been fatal. That some have recovered is shown by the specimens from healed cases. Death usually occurs within one week, and many end fatally in two or three days. The more subacute and chronic cases later on develop peritonitis or metastases.

Treatment.—This is symptomatic, unless a localized abscess is diagnosed, in which case operation offers some hope of success. Mikulicz had a patient thus cured (Lengeman). Bovée¹ recently described an undoubted phlegmonous gastritis cured by gastrostomy and drainage.

Severe Toxic Gastritis.—Various irritant and corrosive poisons when brought into contact with the gastric mucosa induce an intense inflammation which is merely an aggravated form of the acute simple gastritis, the intensity depending on the character of the poison, its nature, concentration, quantity, and length of time present in the stomach, as well as upon the volitional or accidental ingestion of the irritant. These cases are not necessarily severe, and all stages occur from simple hyperæmia to ulceration, gangrene and perforation. Practically all the known poisons may directly or indirectly induce a severe gastritis.

The *exogenous* poisons are especially irritant in their action, *i. e.*, concentrated acids, alkalies and alkaline salts (sodium and potassium salts, potassium and sodium hydrate, ammonia compounds, etc.), metalloids (chlorine, bromine, and iodine preparations, phosphorus, sulphur, arsenic, and antimony), metallic salts (lead, silver, zinc, copper, mercury, etc.), certain of the methane derivatives in large doses (alcohol, petroleum, croton oil, etc.), bodies from the aromatic groups in certain dosages (benzol and its derivatives), ethereal oils, turpentine, balsams; also some of the alkaloids and glucosides, etc.

The *endogenous* group includes poisons which are supposed to result from retention in the body of injurious substances which are under normal conditions eliminated by various channels, *e. g.*, in uræmia and cholæmia, and also poisons resulting directly from metabolic disturbances of a profound nature (diabetes, carcinoma, etc.). Further, there are the bacterial products from all forms of infections, almost any of which may directly or indirectly act as irritants to the gastric mucosa.

The action of the different poisons varies not only according to their nature, but likewise according to the method of ingestion and the position of the victim while taking the irritant. The site of the lesion varies. Some-

¹ *American Journal of the Medical Sciences*, 1908, p. 662.

times but a small portion is affected; sometimes merely the cardia is involved; or again, only the greater curvature. Most change naturally occurs where the poison was longest in contact with the wall, which event occurs in most instances at the fundus, pylorus, and posterior wall.

As repair takes place, scars form, and according to the extent of the original lesion mere cicatrices exist, or else more or less deformity develops. As a result one may find pyloric stenosis, hour-glass contractions, sacculations, etc. Often very extensive lesions heal remarkably well. The mucous membrane is not repaired. The secretory changes are marked. The hydrochloric acid diminishes, or is completely absent.

As a rule, the alkalies are more penetrating than acids; perforation is extremely common, and where the serious effects are not fatal one usually finds more or less deformity of the œsophagus as the result of cicatricial contractions. Often the œsophagus alone is severely affected, for the acid becomes diluted in its journey to the stomach.

Sometimes the lesions in the stomach are characteristic of the kind of poison ingested, *e. g.*, with sulphuric acid the gastric wall externally is much injected, of a dark-blue or violet color, in patches, as though little pockets of fluid had been formed, with marked acid reaction to their contents, and with a coffee-ground appearance. Throughout, the wall is diffusely discolored, much thinned in spots, and here and there perhaps a perforation. The mucosa is gray black in spots or streaks. The ordinary chemical tests applied to the contents can readily confirm the diagnosis. With nitric acid the sloughs have a more yellow color, while the general anatomical features otherwise resemble those of sulphuric acid gastritis. Hydrochloric acid causes a yellowish or grayish color, and the mucous membrane is unusually friable, but deep ulcerations are less common. With copper the discoloration is more of a blue or green, while with silver the mucosa is blackened. Phosphorus induces a milky yellow appearance from fatty degeneration, and carboic acid gives the lining membrane a whitened coating. Lead whitens the mucosa. Metalloids (lead, arsenic, and antimony) are irritants rather than corrosives. So, too, are the metallic salts and methane derivatives (alcohol). Arsenic induces a gastritis, with marked fatty degeneration of the gland cells (gastritis glandularis). With zinc, however, in addition to the ordinary signs, one often finds peculiar, dry, firm sloughs of the mucosa of a whitish color. Iodine and bromine compounds produce an acute gastritis which is no wise pathognomonic of the origin.

Symptoms.—In a typical case the sudden onset of pain and vomiting is the most prominent feature. The onset is severe as well as sudden, the pain is usually felt all the way from the oral cavity to the stomach, and is especially severe behind the sternum and in the epigastrium, and increased by pressure. There are burning pains in the mouth and gullet. Nausea, retching, and vomiting follow almost immediately. The vomiting is frequent, painful, and brings no relief of the symptoms. In the vomitus blood-stained material, perhaps food remnants, mucus, shreds of mucous membrane and the poison itself are found. Great thirst is a prominent symptom, and the dysphagia intensifies the suffering. Diarrhœa appears later, and the stools may show evidences of mucus and blood, usually because of an accompanying enteritis. Signs of collapse rapidly supervene, and prostration, with a cold skin covered with a clammy sweat. The facies is anxious and pale, or cyanosed, and the lips and mouth show evidences of the corroding

poison. Petechiæ often appear in the skin, and jaundice is not infrequently present from toxic or obstructive causes. The pulse is rapid, small, and often irregular in volume and rhythm. The respirations are shallow, quickened, and thoracic in type, because the diaphragmatic movements are limited through the pain. The temperature, at first low, soon rises above the normal, varying according to the anatomical changes, the general toxic results, and the general state of the patient. The condition of the sensorium varies according to the nature of the exciting cause. In many instances there is great excitement, convulsions supervene, and, later on, coma.

The urine may show the effects of a toxic nephritis, or may be unchanged, the condition depending on the kind of poison ingested. The abdomen in the acute stages shows nothing beyond tenderness and distention, unless perforation and peritonitis have occurred with their usual signs. Death occurs either from exhaustion, or convulsions, or suffocation, or from general intoxication. Should recovery take place, complete restoration of the tissues may occur in those cases where no marked corrosion of the gastric mucosa has been present, but this is only of course with the milder forms of irritant poisoning. In other cases the results are less favorable. Chronic indigestion supervenes from atrophy of the glands, or persisting gastritis with vomiting, progressive weakness, and emaciation. Sometimes ulcerations persist, or their healing results in the formation of stenoses and deformities.

Diagnosis.—This is based on the history, the nature of the symptoms, the oral and pharyngeal evidences of corrosion, etc., the odor of the breath in certain cases, the chemical analysis of the vomitus or of the washings from the stomach, and the analysis of the urine.

Prognosis.—This depends upon the special cause, the kind of poison, its amount in relation to its toxicity and irritant properties, the early emesis, and the possibility of early removal. Collapse and peritonitis are serious symptoms.

Treatment.—Rational therapeutics depends upon a recognition of the special toxic agent, for which the proper antidote can be given. In addition to this, one should attempt to get rid of the poison, if early recognized, by rapidly acting emetics, *e. g.*, apomorphine. Lavage should be used when it is improbable that extensive corrosion is present, and soothing remedies for the gastric mucosa may then be administered. For corrosive acids one may give mucilaginous and oily drinks or weak alkalies. For corrosive alkalies, vinegar and water, or citric acid are useful. Counterirritants externally may aid in giving relief, while medicinally one should use demulcents, opiates, bismuth, etc. Rectal feeding will be necessary for some days. Collapse must be checked or treated by appropriate remedies.

Mycotic Gastritis.—The human body fluids, owing to their deficiency in free oxygen and their alkaline reaction, are for the most part unsuitable media for the development of most of the moulds and yeasts, which thrive better on dead organic material. A few, however, and notably the actinomyces, occupy a special place in pathogenicity, and some others occur not infrequently either as a cause of disease or associated with maladies due to other agencies. Many of the commoner parasites are found in the stomach under normal or abnormal conditions, both fission fungi (bacteria) and mould fungi, as well as yeasts, and while in many instances they are the direct cause of changes in the gastric functions or anatomical conditions, at other times their presence seems to be rather accidental and unimportant.

Inflammation of the gastric mucous membrane as a result of bacterial invasion through the blood or lymph streams is common enough, and gastritis due to streptococci, typhoid bacilli, pneumococci, and other infectious agents is well recognized. Whether or not bacteria which are ingested with the food can induce a true acute gastritis seems still open to question. Kellogg,¹ after numerous analyses of gastric contents, concluded that ingested bacteria when very numerous might very probably be the cause of acute inflammation, although others have contested his views. It would appear that the acid gastric juice retards the growth and development of bacteria, although moulds seem to resist the acid better than do the schizomycetes, which latter grow better when subacidity is present. The acidity, the stagnation, and the quality of food ingested are the factors which determine the growth or retardation of development of the various fungi in the stomach.

As a general rule, moulds and yeasts do but little harm in the stomach, and do not usually produce anatomical changes, or prevent recovery from various forms of gastric disease. Kundrat² found favus in the stomach associated with a favus universalis, which was distributed throughout the body, and Rudnew³ found the penicillium glaucum in a woman who died of Asiatic cholera.

The presence of moulds and yeasts seems to have an anatomical rather than a clinical interest and importance, although certain signs and symptoms are significant to the physician. Rarely they are so intimately associated with anatomical lesions (inflammation of the mucosa, erosions, ulceration, etc.) as to render it highly probable that sometimes they may cause or aggravate these maladies. De Bary found various moulds in seventeen patients suffering from gastric disorders of different kinds—among others mucor mycelia, leptothrix buccalis, thread fungi, and oïdium lactis—as well as others of undetermined species.

Klebs and Fraenkel associated a gastritis with favus, yeast, and oïdium albicans; certainly the last-named fungus has often been seen in cases of gastritis, and yet in spite of its frequency during childhood in the pharynx and œsophagus, it is rare in the stomach. Epstein drew attention to the fact that a healthy mucosa was not a suitable medium for the growth of this parasite in the pharynx, and perhaps the same applies to its growth in the stomach. This may account for the frequently abrupt termination of the affection at the cardia, despite the fact that large numbers of the fungi reach the interior of the stomach.

Rare as this affection is in children, it is still more infrequent in the adult stomach, and then occurs only in connection with some debilitating condition or metabolic disturbance. In a case described by Maresch⁴ the oïdium albicans, originally in small numbers on the surface of an œsophageal carcinoma, had nevertheless induced an extensive growth of the parasite in the stomach, this being associated with an ulcerative gastritis. The fungus grew not merely on the surface, but deep down as far as the muscular layer. Apparently the gastric inflammation and ulceration formed a satisfactory nidus for the development of the parasite. The anatomical appearances were characteristic. In various parts of the stomach there were several yellowish and brownish, sharply defined areas, flat, slightly projecting beyond the surface

¹ *Medical News*, July 21, 1900.

³ *Virchow-Hirsch Jahresh.*, 1867.

² *Wien. med. Blätter*, 1882, No. 49.

⁴ *Ztschrft. f. Heilkunde.*, April, 1907.

of the mucosa, to which they were rather firmly adherent. The central portions were slightly depressed and darker than the periphery. Perpendicular section through these plaques revealed the fact that their under surface was intimately connected with the deeper layers of the submucosa.

Reisz had a similar experience in which the stomach of a girl aged eighteen years was covered with patches of fungus growth, the underlying epithelium was partially degenerated, and the mucosa infiltrated with small round cells. Bastedo described a case in which the thrush had apparently induced gastritis in an elderly woman, and in which the removal of the supposed cause by lavage was rapidly followed by recovery.

Von Wahl¹ and v. Recklinghausen² recorded cases where fungi of the lepto-thrix group penetrated the gland ducts and were anatomically associated with necrotic areas. In one case many pustules existed in the glandular layer and submucosa of the pylorus, in the other the fundus was chiefly involved. Virchow doubted the etiological relation between these moulds and the anatomical lesions, regarding their presence rather as a cadaveric phenomenon, while Klebs, on the other hand, believes in their pathogenicity.

If, however, moulds do sometimes create inflammatory disturbances and abscess formation, the instances are undoubtedly rare, for moulds are usually soon expelled through the pylorus, do not remain long in the stomach, and merely induce disturbances of function when present in large numbers.

Symptoms.—The relation of the presence of moulds to the symptoms is dubious. Talma was of the opinion that the carbohydrate fermentation which was caused by the presence of microorganisms, was the antecedent of many cases of hyperacidity, but although moulds may co-exist with hyperacidity and gastralgia, it is not certain whether their presence is a cause or an effect of the condition found.

Einhorn³ has admirably described a few cases, and was among the first to recognize the condition clinically. The lavage water from the empty stomach demonstrated the presence of blackish-gray or brownish-green flakes indicating the existence of moulds which microscopically showed spores and mycelia. The species could not be determined even though carefully examined. These flakes varied in number from 4 to 50 or more, were 2 to 5 mm. in size, and often intimately mixed with mucus, which fact together with their presence in the empty stomach argues that their presence is not merely an accidental admixture. Probably the moulds adhered closely to the mucous membrane and perhaps proliferated in it. At all events they can evidently thrive in the stomach and retain their power of growth, regardless of the gastric juice.

Two types of cases occur, one with signs of hyperchlorhydria (occasionally with hypersecretion and vomiting), the other with gastralgia, the gastric secretion being normal or reduced. The relation of the fungi to the symptoms is either etiological or a cause of increased severity. Lavage improves the symptoms.

Treatment.—Systematic lavage rapidly removes the fungi permanently from the stomach. Einhorn advised the further use of weak solutions of silver nitrate in the lavage water.

Diphtheritic Gastritis (Gastritis Membranacea).—This condition, which has much more anatomical than clinical significance, may be defined

¹ *Virchow's Archiv*, 1861, xxi p. 579.

² *Ibid.*, 1864, xxx.

³ *Medical Record*, 1894.

as a pseudomembranous inflammation of the gastric mucosa, usually a sequel to a similar condition of the nasopharynx, but sometimes primary (Delafield) and associated with varying bacterial agents, and at other times apparently due chiefly to chemical causes.

True diphtheria of the stomach, due to the Klebs-Loeffler bacillus, is a rare condition and nearly always secondary to a similar bacterial invasion of the contiguous parts, œsophagus, throat, or upper respiratory passages. Andral saw such a case in a girl, aged ten years, in whose stomach there were long and broad strips of diphtheritic membrane evidently associated with false membranes of the same character in the pharynx and œsophagus. In Vidal's case of similar origin there were fine small areas along the greater curvature, while Steiner and Neureutter had a similar specimen showing the chief involvement at the lesser curvature.

Kalmus was of the opinion that 6.5 per cent. of his cases of diphtheria showed secondary pseudomembranous lesions in the stomach, but no other writer has confirmed his observations as to its great frequency. Leary had observed only two instances out of 136 fatal diphtheria cases examined at the Boston City Hospital.

While, as a rule, diphtheritic gastritis is secondary to some other diphtherial infection, it may complicate various other infectious diseases, *e. g.*, typhoid fever (Thorel¹), pneumonia (Foulerton²), pyæmia, the exanthemata, etc. Isolated instances of the malady have been described by Sir William Jenner, Wilson Fox, Wilks, and others. Children seem to be most commonly affected, and with them, apart from the diphtherial infection as a cause, tuberculosis is frequently the fatal primary disease. Cahn described from Kussmaul's clinic an instance of diphtheritic gastritis associated with acute yellow atrophy of the liver. A true pharyngeal diphtheria developed, and with it a secondary diphtheritic gastritis, while the liver condition seemed to be a complicating incident.

The experiments of Enriquez and Hallion,³ where necrotic changes with ulceration occurred in the gastric mucosæ of dogs and guinea-pigs, after injections of diphtheritic toxins, have, of course, no relation to the condition commonly known as diphtheritic gastritis—their results being merely such as might occur in any general mycotic or toxic infection; some simple chemical poisons produce similar effects, *e. g.*, ammonia.

Morbid Anatomy.—With a Klebs-Loeffler infection the pharynx is usually involved, so also the œsophagus, although the latter may be passed over, and the membrane evidently swallowed without involving the gullet on the way. The stomach is not usually altered in size or shape. The chief feature is the presence of the pseudomembrane. This may be diffuse and cover the entire surface of the stomach, as in the case described by Talfourd Jones,⁴ and that by W. S. Fenwick,⁵ but more often there are streaks or patches of membrane—sometimes radiating from the cardia toward the pylorus, or in isolated patches of varying size, thickness, and number, situated in the fundus, along the curvatures or in different places, and irregularly arranged. The mucosa is discolored, usually darker than

¹ Lubarsch and Ostertag, *Allgem. Path.*, 1898, vol. v, p. 156.

² *Transactions of the Pathological Society of London*, 1902, p. 286.

³ *Compte. Rendu. Hebd.*, 1893, p. 1025.

⁴ *British Medical Journal*, 1889, i, p. 880.

⁵ *Transactions of the Pathological Society of London*, 1894, xlv.

normal, reddish or grayish red in appearance, and thickened. The rugæ are usually well-marked, and covered with the more or less firmly adherent membrane.

Microscopically the picture resembles that seen in ordinary pseudomembranous lesions of any mucous membrane. The underlying epithelium is more or less destroyed, the lymphoid tissues are sometimes increased, and a coagulation necrosis forms the superficial layer. Leary noted the concurrence of streptococci in the only two instances which came under his notice among the fatal diphtheria cases with pseudomembranous gastritis. Some have found tubercle bacilli as a coincident infection. As in pseudomembranous inflammations elsewhere, so here the bacteriological findings vary widely. Streptococci and Klebs-Loeffer bacilli are those most commonly isolated.

Symptoms.—As has already been said, the condition has more anatomical than clinical interest. There are no characteristic symptoms, and the mere vomiting of a pseudomembrane does not necessarily imply that the disease has affected the stomach itself—unless, indeed, a cast of the whole stomach be vomited up, as was recorded by Thomson¹ in one instance. Such membranes are readily swallowed and sometimes are so extensive as to cause pyloric obstruction. Seitz,² however, has recorded a diagnosis of pseudomembranous gastritis during life and confirmed at autopsy. Some of the membrane had been vomited concurrently with the presence of other evidences of gastric disturbance. As a rule, however, the origin of the membrane is not easy to determine by clinical methods, and its presence in the stomach may have been merely an accidental dislodgement from the pharynx, œsophagus, or respiratory tract.

CHRONIC GASTRITIS.

Definition.—Chronic gastritis consists in a protracted inflammation of the gastric mucous membrane resulting in changes in gastric digestion. These changes may be in the secretions, in the motor power, or in both, and, according to the type of the disease, the symptoms and signs may differ widely. It may be primary or secondary.

Incidence.—In its primary pure form the condition is clinically quite uncommon—at all events much more rare than is ordinarily supposed—being frequently mistaken for a purely functional disease in which similar signs and symptoms exist without any pathological changes in the gastric mucosa. For this reason, and in view of the doubt frequently existing in the absence of more accurate means of diagnosis, the time-honored term “chronic dyspepsia” should not be despised. It may be that after some time even functional disturbances lead to pathological changes in the gastric mucous membrane, a true superficial inflammation, as in the cases referred to by Cohnheim, in which the gastritis resulted in his opinion from motor insufficiency, and was induced by irritation of the stagnant chyme. However, the conception as to what constitutes a gastritis clinically has been a subject of controversy for many years and seems still to be a matter of doubt in the minds of many writers. Faber, who found small-celled infiltration of the

¹ *Archives of Pediatrics*, April, 1895.

² *Diphtheria and Croup*, 1877.

mucosa very common with diminished acid secretion, deals with a pathological rather than with a clinical state.

As a primary affection, chronic gastritis is not often found in the autopsy room, the cause of death usually being some general or local condition to which the gastritis is secondary (cardiac or hepatic disease, nephritis, tuberculosis, pernicious anæmia, etc.). Thus, in the Royal Victoria Hospital, among 1300 autopsies, there were 108 cases with chronic gastritis, and of these, 92 were definitely secondary to other causes, while the remaining 16 were more or less mild in type and associated with other diseases not necessarily the cause of the condition. Statistics, however, with reference to the incidence of chronic gastritis are notoriously unreliable.

Clinically, much doubt still exists as to the accuracy with which various signs and symptoms may be considered as evidence of true inflammation of the stomach, and in this way much has been called gastritis which undoubtedly would more truly come under the category of neurosis of the stomach, or under the old name of chronic dyspepsia, a purely functional disorder often due to atony. Vice versa, many cases of chronic gastritis as seen at autopsy (secondary to other diseases) were utterly devoid of clinical evidence of that malady during life.

Pathologically, statistics are almost equally unreliable, through the infrequency with which primary gastritis comes to the pathologist's notice and because of the numerous errors in pathological observations on this condition. Marked contractions of the gastric wall are mistaken for chronic thickening, and passive congestion for true gastritis, where a microscopic examination would have revealed no sign of disease. In the same way, too, the so-called polypoid excrescences are regarded by many as signs of chronic gastritis when often they would with more justice be placed among hypertrophic conditions or neoplasms, showing as they often do no microscopic evidences of true inflammation. Efforts to overcome these difficulties in accurate diagnosis have recently been made, and with some success, by Faber and others. The rapid postmortem changes and the persistence of autodigestion have been partially prevented by the introduction into the stomach of 10 per cent. formol solution immediately after death. In this way better specimens of the mucosa as it existed in life have been obtained, and the relation of symptoms and signs to pathological changes have been determined with somewhat greater accuracy. But much remains yet to elucidate the conditions associated with this malady, and to explain the reasons for the presence or absence of many of its symptoms.

Classification.—Clinically this can scarcely be said to have been satisfactorily determined. Classifications have been based upon the nature of secretions, and thus gastritis *acida*, gastritis *anacida*, gastritis *mucosa*, etc., have been described. The mere analysis of the secretions, however, affords practically little clue to the underlying conditions, and per se cannot, in many cases, even determine differentiations from certain neuroses. In many instances a hyperacid secretion is found in the early stages of chronic gastritis to be followed later by a diminution of the hydrochloric acid. Sometimes, however, hyperacidity may appear late, while not infrequently it persists throughout the whole course of the affection.

When one realizes how widely the secretions may vary from day to day; how on one day an excessive acidity may be followed by a marked hypochlorhydria on the next; how, moreover, there are times of great mucous

secretion with rapidly following cessation of it, one can scarcely feel safe in relying upon a classification of chronic gastritis based on a state of affairs which may be either ephemeral or of great variation from time to time. As well might one classify the various forms of pneumonia according to the amount and character of the sputum.

The term mucous gastritis, for example, is used to designate the simple chronic gastritis, or gastric catarrh, in spite of the fact that mucus may be abundant in any of the different pathological varieties of this malady. Again, in hyperacid gastritis much or little mucus may be present, and the underlying condition may be proliferative and degenerative in nature. Proliferative gastritis, as a rule, induces diminution in the secretion of HCl, although, in many instances, there is hyperacidity. In cases of chronic gastritis with atrophy of the glands, anadenia—achylia gastrica—the symptoms and signs are apt to be most constant; in certain instances of this variety there is a rapid course with signs of chronic gastritis and progressively downward tendency. Examination of the gastric contents after a test meal shows little or no digestion, and the secretions appear absent. Here, however, as in the other forms of chronic gastritis, great variations occur in the symptoms at times, and, moreover, there are all grades of transition to the other varieties of the disease. Again, whether owing to treatment or to the natural course of the malady, a hyperacid gastritis may change to the anacid type, and vice versa; if enough gland elements remain, the condition of achylia gastrica may alter to one with normal or even hyperacid secretion.

Apparently the state of the motor power has greater significance in the production of clinical signs and symptoms than has the secretory function, and many have described the persistent evidences of the malady as only becoming marked when there is motor insufficiency. Certainly many cases in which autopsy has shown marked disease of the gastric mucosa have given few symptoms during life so long as the motor power was unimpaired. It would seem well, therefore, to avoid any clinical classification into subdivisions, and to recognize the fact that the manifestations being varied, one may recognize the malady from the complexity of signs, symptoms, etiological factors, and course, and be content with the general diagnosis in all such cases of "chronic gastritis."

Etiology.—Primary chronic gastritis is mainly caused by irritants to the gastric mucosa, acting over a prolonged period and in a strength not so great as to corrode. Oft-recurring acute gastritis induces the chronic form. Alcohol is the commonest irritant and the most important cause, especially if taken undiluted, *e. g.*, brandy, gin, wines of all kinds when taken to excess—their action being either as direct irritants or perhaps in part due to the production of acids in the stomach. So also diluted alcohol may act in the same way by inducing acetic fermentation.

An analysis of cases at the Royal Victoria Hospital, however, has called attention to the fact that alcohol in excess is by no means a necessary cause. In 10 autopsies performed on men dead of acute alcoholism, 7 showed no definite chronic gastritis whatever, while in the others the signs were marked in only one. Of the autopsies upon 40 other patients, in whom a marked alcoholic history was obtained, but in whom death was due to other causes, there were 20 cases with chronic gastritis, and the remainder showed no definite signs of true inflammation; 10 showed mere congestion, 10 showed

swelling and congestion, 9 showed a granular mucosa, and only 5 showed definite thickening; 4 others had mammillations, and true polypi existed in the remaining 2. Macroscopic erosions existed in only 1 of these 40 cases. It is interesting to note that 21 of these 40 patients gave no history of gastric symptoms, while 16 had pain and vomiting.

Other causes of irritation acting directly or indirectly are spices, strong tea or coffee, certain irritant drugs and drastic purgatives, as also tobacco, especially if chewed. Dietetic errors, if maintained over a sufficiently long period are an important cause, *e. g.*, the improper quality of food (if coarse, too rich, too spicy, fatty, or otherwise irritating), excessive quantity, as regards bulk, especially of rich foods, improper mastication of food and also irregularity at meals. All these are of greater importance in proportion as the individual's habits are more sedentary.

Secondary gastritis may be due to local or general conditions. The chief local conditions are as follows: Gastric diseases of any chronic nature, affections of the portal system, especially obstruction, and also the presence of parasites (bothriocephalus, etc.). The main general conditions are numerous, *e. g.*, cardiac and arterial disease, pernicious anæmia, leukæmia, gout, diabetes, etc., and general infections (typhoid fever, pneumonia, tuberculosis, sepsis, etc.).

Morbid Anatomy.—Pathologically two main types of chronic gastritis exist, the one *chronic productive*, the other *atrophic*. In the productive type, part or all of the stomach wall is thickened, while in the atrophic variety it is thinned in one or all of its coats. The stomach is usually larger than normal, except in the cirrhotic forms where there is shrinkage of the organ. Its shape is variable. At autopsy, mucus is usually present in the gastric contents in more or less abundance; it is usually thick, of a more or less grayish or slaty color, and sometimes reddened with blood pigment. It is either translucent or opaque, and sometimes appears semipurulent.

The mucous membrane varies according to the type of gastritis present. In the *productive variety* the mucosa is swollen and thickened, and of a dark-red or grayish-red color, especially in the neighborhood of the pylorus. The color varies according as there is distention of the vessels, diapedesis, or small hemorrhages, and further according to the duration of the malady. In the older cases there is more pigment, which appears beneath the mucosa as a black or dark-gray discrete discoloration. Not infrequently one sees either small, follicular ulcers or hemorrhagic erosions, with destruction of epithelium and loss of substance in the mucous membrane. Such ulcers have usually well-defined edges, are superficial, crater-like, and apparently due to inflammatory hyperplasia of the lymph follicles, with subsequent degeneration. The affection of the mucosa is either general or local. Where it is diffuse one gets a dense thickening from interglandular inflammation as well as from productive inflammation of the gland structures. The thickened wrinkled mucosa with its flat mammillated appearance may be so markedly changed as to give an almost polypoid appearance to the individual bones, and to this latter condition the term "gastritis polyposa" has been given. It is, however, essentially different from the condition of the gastric wall in which a few simple polyps are found apart from any chronic inflammation. The term "gastritis granulosa" designates a diffuse productive inflammation where the mucosa is thickened and the appearance abnormally granular.

PLATE VII

FIG 1



Chronic Productive Gastritis.

FIG. 2



Chronic Productive Gastritis.

PLATE VIII

FIG. 1



Chronic Productive Gastritis.

FIG. 2



Chronic Atrophic Gastritis.

The submucosa and muscularis may also be affected, the muscle itself thickened, and the interstitial tissue increased. One may even see the serosa involved.

Microscopically, the mucus overlying the gastric epithelium usually contains glandular epithelium more or less degenerated, and perhaps some blood corpuscles and pigment. The mucous membrane itself shows mainly small-celled infiltration, degenerated epithelium, hemorrhages, cystic dilatation of the gland tubules, and here and there some true erosions. The round-celled infiltration occurs early, is usually chiefly superficial and beneath the uppermost layers of epithelium, as also to some extent between the glands. Its arrangement may be diffuse, in striæ or in clusters simulating enlargement of the isolated lymphoid follicles. True fibrous tissue formation is comparatively uncommon, and there is little tendency to sclerosis. Small round-celled infiltration may persist for a long period of time without developing into fibrosis. The degeneration of epithelium affects mainly the secreting cells, while the mucous cells appear to resist the inflammation longest.

Often the epithelium is so distorted that it is impossible to distinguish chief from parietal cells, and many assume a cylindrical or a cuboidal shape. Cystic dilatation of the tubules is common (retention cysts). This so-called "gastritis cystica" is sometimes very pronounced, and one may observe on careful examination that the cysts are very numerous, sometimes covering almost the entire surface of the inner wall. Capillaries and lymphatics are usually dilated. Here and there are hemorrhages, old and new.

Atrophic Type.—The second type of chronic gastritis, with atrophy of the mucous membrane, sometimes called *anadenia ventriculi*, is said to be preceded usually by gastritis proliferans. The mucous membrane is devoid of properly developed glands. The surface is pale, thin, and smooth, or perhaps slightly reddened from hemorrhages. In the advanced cases the surface consists more of granulation tissue or well-developed fibrous tissue. The muscularis takes part in the process and is usually atrophic. Sometimes, however, there is muscular hypertrophy with atrophy of the mucosa, and one may see pigmentary changes in the latter, the so-called atrophic pigment induration of the stomach.

Microscopically the mucosa resembles more and more that of the small intestine. There are distinct evidences of round-celled infiltration, not only in the superficial layers but in the deepest layers of the glandular structures. The atrophy of gland structures varies; often the secreting glands are alone involved, while the mucoid cells are unaffected; but the changes in this respect vary in different cases, as well as in different portions of the stomach in each individual instance. Illustrative types of these cases are often found in pernicious anæmia. Some advanced cases are difficult to distinguish from a diffuse scirrhus, the gland structures being so atrophied that only a flat fibroid-looking surface remains whose base encloses the remains of gland structures.

Cirrhosis of the stomach is only another variety of the atrophic in the sense that gland structures disappear. Here the organ is smaller than normal, contracted, and thickened usually in all its coats from excessive growth of the products of chronic inflammation.

Symptoms.—The disease, both in its primary and secondary forms, may remain latent throughout or for a long period, or the symptoms may be at first insignificant and only gradually become more pronounced. To a certain extent the symptoms and signs depend upon the condition of the secretions and motor power, although certain general features apply to most of the cases. It is, at all events, well recognized that the symptoms are often most variable.

Early symptoms are usually those of a so-called dyspepsia; slight dietetic errors cause discomfort in the epigastrium, with a sense of fulness, pressure, or weight, and perhaps even slight pain, either while the stomach is full or when the food has passed on into the intestine. The pain, however, is not, as a rule, severe, or sufficient to interfere with the individual's daily routine. Unless hyperacidity exists there is usually anorexia, or the appetite is fickle and yearning for spices, acids, or liquids, with a distaste for meats, is common. A disagreeable taste in the mouth is present, and flatulence commonly supervenes, often being a marked symptom. Eructations of gas frequently relieve the distress. The tongue may be furred and is often sore at the edges and tip. Nausea and vomiting occur either at the height of digestion or sometimes in the early morning—perhaps induced by the swallowing of pharyngeal mucus during the sleep. Thirst is usually present, although less common than in acute cases. Lassitude, neurasthenia, and dizziness during the digestive period are the rule. Later in the disease, when the condition is more pronounced the symptoms are aggravated. Nausea is more persistent, waterbrash and cardialgia are more common, and vomiting comes on with greater frequency and regularity according as there are indiscretions in the diet. Fluids may be well borne, but solids aggravate the symptoms. The vomiting, which now often occurs in the morning, consists largely of mucus, and is often alkaline in reaction. If emesis occurs after meals, the food is seen to be but partially digested, and mixed with thick, ropy mucus, bile, and the various products of fermentation.

The intestines are likewise affected, and constipation is the rule, although sometimes this alternates with diarrhoea, or, again, the latter may be the chief evidence of the original gastric inflammation.

Nervous symptoms are frequent, especially neurasthenia, hypochondriasis, inertia, dizziness, palpitation, etc. The nutrition varies, and usually there are signs of inanition, especially when due regard is not paid to the treatment of the mouth and digestive tract generally. Emaciation may be rapid when the intestinal functions are simultaneously impaired. The urine varies. Urates are usually abundant in the dark, concentrated specimens, and calcium oxalates are often in excess. Not infrequently the ethereal sulphates are much increased.

Physical examination of the abdomen gives little information, as the only constant sign is tenderness—more or less diffuse and perhaps more often found to the right of the epigastrium than elsewhere. The stomach may or may not be distended, and there is no visible peristalsis, as a rule, unless pyloric obstruction exists.

Gastric Contents.—Usually the food removed is abnormally large in amount, appears undigested, coarse, and mixed with mucus. The most constant abnormality is the excessive amount of *mucus*, and the quantity is best judged by the macroscopic appearance. Its presence, however,

does not imply a disturbance of gastric function, if the origin be, as is frequently the case, in some portion of the tract above the stomach, *e. g.*, pharynx, œsophagus, or from the bronchi, larynx, etc.

Mucus from the stomach occurs more commonly in shreds or flakes, not in isolated lumps, is mixed with the food, and is usually heavier than that from the respiratory tract, which contains air and therefore floats in water. The mucus from chronic gastritis is often found in most abundance after the test meal has been removed and the remaining contents gained by thorough lavage. It usually contains leukocytes in more or less abundance, as distinguished from the mucus of the respiratory tract, which shows epithelial cells and perhaps pigment. The mucus is most abundant in those cases of gastritis with atrophy of the gland structures and is least common in hyperacid conditions, although even to this rule there are many exceptions. The presence of abundant gastric mucus enclosing many leukocytes is positive evidence of gastric catarrh.

The *secretion of acid* varies. Hyperacidity is uncommon, and when present usually indicates an early condition of the gastritis, which may account for infrequency with which it is found. This, when present with much mucus and many leukocytes, affords evidence of the so-called gastritis *acida*. Diminution of hydrochloric acid is more common and may be progressive up to the period of total achylia (*achylia gastrica*, atrophic gastritis).

From time to time, however, during the course the amount of acid shows great variation. McCaskey, in an analysis of 600 cases of so-called chronic gastritis, found no hydrochloric acid in 20 per cent., subacidity in 26 per cent., normal acidity in 34 per cent., and hyperacidity in 20 per cent. The *pepsin* keeps pace with the hydrochloric acid, and the *rennet ferment* likewise diminishes with the progress of the disease. Bouveret regards a quantitative estimation of the rennet ferment as a valuable aid in prognosis as indicating possibilities of repair.

Achylia not infrequently occurs in the late stages of chronic gastritis, and supervenes upon a progressively diminishing secretion (gastritis atrophicans). From the pathological standpoint achylia does not imply anadenia. Faber especially draws attention to the fact that in gastritis there may be achylia even although many secreting cells are unimpaired, and concludes that the accompanying gastritis has merely disturbed their function but not their anatomical structure. In order to prove achylia several test meals should be given, inasmuch as numerous conditions may induce a temporary absence of gastric juice. The enzymes are absent as well as the hydrochloric acid; mucus may be abundant.

Organic acids are not present except in small amounts, as a rule, and only if there be stagnation of food. Lactic acid is not usually found where a test meal has been given after a previously thorough lavage.

The *motor power* varies. As a rule, it is not greatly impaired, although if insufficiency be present there is much more fermentation and the general gastric symptoms are much more pronounced. In most cases of typical chronic gastritis there is some atony, more readily induced by persistent dietetic errors. The gastric functions are readily taken on by the intestines where normal motor power exists. Gastrectasis is uncommon.

Some authors (Cohnheim¹ and others) describe valuable aid from examina-

¹ Arch. f. Verdauungskrank, 1896, i, p. 238.

tion of *bits of mucous membrane* which sometimes come away with the lavage water, but Lubarsch's¹ studies have revealed the many sources of error liable to result from trusting too implicitly to this means of diagnosis. The alterations they present are often but semblances of inflammatory change, and vice versa, even if the cells and glands be actually altered structurally, it does not necessarily mean that they have been functionally inactive.

Course and Prognosis.—It is usually of long duration, and the general health suffers either periodically or progressively, but alternation of good and depressed health is the rule. The mental condition suffers, neuroses appear and often great physical depression as a result of the hypochondriasis and neurasthenia. The exacerbations usually depend on indiscretions, and recurrences are very common, especially in alcoholics. The mild cases are readily cured, but in the more severe ones the probability of cure depends on the condition of the muscular wall, the state of the mucosa, and the kind and degree of inflammation present.

With atony of the stomach, and especially gastrectasis, and with achylia gastrica, the condition is much more difficult to treat successfully. When the intestines are healthy, the chances of cure are naturally augmented. The prognosis should be based less upon the general condition than upon the outlook for securing a sufficient supply of nourishing food and a maintenance of secretory and motor functions. In every case the individual affected has himself most to do with the cure by observing due regard to the requirements of treatment, which, after all, are readily followed and are usually of benefit in cases not too far advanced.

Diagnosis.—The condition is very often latent, and repeated observations may be necessary; the symptoms and signs as above described, with the recognized etiology, render the diagnosis certain from exclusion rather than from any other means. The main features in the diagnosis are: (1) The etiological factors. (2) Persistent indigestion, with nausea and frequent vomiting of much mucus, gastric pain, and tenderness. (3) The relation of the symptoms to the ingestion of food. (4) The variations in the course, with its exacerbations and remissions. (5) The moderate loss of strength and weight as compared to the lowered nutrition in more serious conditions. (6) The comparatively slight alterations on analysis of the motor power. (7) The presence in the gastric secretions of much mucus and variable quantities of acid from complete achylia to hyperacidity. (8) The finding in the stomach contents of many leukocytes either in the mucus or in bits of mucous membrane coming away in the washing. (9) The beneficial effect of proper treatment.

Differential Diagnosis.—One must distinguish between primary and secondary forms, the latter which usually accompany other diseases being readily recognized by detection of the original cause.

Chronic Gastritis and Gastric Neuroses.—The differentiation is often impossible, for in each case there may be evidences suggestive of the one malady, and yet the course and other factors may indicate with some degree of certainty that the other disease is present. The distinctive features are as follows: In chronic gastritis: (1) The cause is usually obvious. (2) The condition of the patient is poorer and the nutrition is apt to suffer more. There is, moreover, less evidence of neuroses else-

¹ *Achylia Gastrica*, Leipsic, 1897.

where. (3) The symptoms occur during the digestive period, and are dependent upon food, its quantity, quality, and time of ingestion. There is vomiting of much mucus. (4) The analysis of the contents shows much mucus, which often contains many leukocytes and the same may be found in bits of mucous membrane which may have come away with the washings. There is but little disturbance of motor power and the secretions are variable from complete achylia to hyperacidity (all of which results may, however, be found in neuroses). (5) Examination for enzymes shows that their presence or absence keeps pace with that of the hydrochloric acid, whereas in neuroses there may be absence of hydrochloric acid, but enzymes are then usually present. (6) The course is with exacerbations and remissions lasting over weeks or months, while in neuroses the intervals of change are much shorter and more variable. In the latter, days of apparently complete health alternate with those of great mental and physical depression.

Gastritis versus Cancer of the Stomach before Tumor is Evident.—In the early stages this is often difficult and usually impossible at first. In many cases the symptoms may justly be referred to either disease, and development alone will determine the diagnosis. In gastritis the onset is usually insidious, from a well-recognized cause, and there are exacerbations and remissions, usually associated with dietetic errors. In cancer the disease, while often insidious at the onset, progresses when the gastric digestion is still *apparently* good; a disturbance appears without cause, persists, and progresses in spite of all care. In gastritis the nutrition suffers less; there is no cachexia or great wasting; the pain is mild; no fever exists. There is vomiting, although less frequently than in cancer, especially when the cause is removed, and hæmatemesis is much less common. In gastritis the gastric analysis shows much less motor insufficiency, a variable amount of hydrochloric acid, and rarely any lactic acid. The reverse holds for cancer. The course of the disease in gastritis is less rapid, with frequent remissions, and treatment has a beneficial effect upon all the symptoms. Later, exclusion aids the differentiation, and in the absence of downward progress and tumor or metastases the diagnosis of gastritis is strengthened.

Atrophy of the gastric follicles, achylia gastrica, is an occasional sequel of chronic gastritis, as also an accompaniment of gastric cancer, pernicious anæmia, etc. It may, however, occur as a neurosis. Thus, Ewald, Einhorn, and others have found that for years there may be complete absence of hydrochloric acid in the gastric secretions in individuals who are either quite well or merely neurotic. These patients show no dyspeptic signs in spite of the hydrochloric acid deficiency, and, moreover, show a mucosa which is perfectly normal according to observations made upon bits of mucosa removed in the washings. In many instances even after years of achylia the secretions may return to the normal.

The presence of much mucus in the gastric contents in cases of achylia is one of the chief determining factors of the diagnosis in favor of gastritis, implying, as it does, a probable catarrh; and this will be strengthened by the finding of a number of leukocytes in the flakes of mucus removed.

Treatment.—A detailed diagnosis is essential to the proper treatment of chronic gastritis. *Secondary* gastritis must be treated according to the cause, which will involve the therapeutics of the lungs, heart, liver, or general constitution. In the *primary* cases, on the other hand, the treatment em-

braces prophylaxis, palliative and curative measures. The curative measures are mechanical, dietetic, and medicinal.

One must consider the cause of the disease as well as the condition of the secretory and motor functions and the quantity of mucus present.

Prophylactic.—Prophylaxis is of some importance, and one should, therefore, remove any external conditions which tend to bring on or aggravate the malady. Excesses of all kinds must be guarded against; bad habits cured; the patient should be taught to eat slowly, to chew his food well, and carefully to select his diet both as regards quantity and quality. Faulty teeth, as forming an important factor in the etiology of gastritis, should be repaired. The actual treatment is chiefly mechanical and dietetic, while drugs play a comparatively unimportant part.

Mechanical.—The mechanical treatment consists mainly in lavage. It is necessary that the stomach should be clean before any food enters, and for this reason one must remove any mucus which covers up the superficial epithelium and prevents proper action of the digestive juices. Lavage may be carried out in the early morning, or, in bad cases, six hours after dinner (which is usually taken at midday), and the evening meal should be as light as possible. Under ordinary conditions one may wash the stomach out with simple lukewarm water. If, on the other hand, much mucus is present, sodium chloride may be added, or, if there be much fermentation, boracic acid; according to the severity of the case, lavage should be done daily or every second day, for a few weeks, and on each occasion it should be continued until the water returns quite clear. When atony or much fermentation is present this may require some practice.

When, for one reason or another, lavage is impossible, one may employ "natural lavage" by means of frequent administration of mineral waters of various kinds, especially the saline waters with carbonic acid, which are often beneficial in relieving the stomach of its mucus and inducing a combination of the organic acids. The alkaline sulphates, such as are combined in Carlsbad waters, are useful for this purpose as well as for relieving the constipation. Where, on the other hand, there is irritation in the stomach with diarrhoea, they are best taken hot. Whenever atony exists it is well to remember that only small quantities of fluid should be taken at a time.

Daily cold baths or shower baths, with subsequent friction of the skin, are of great benefit. Where pain or great discomfort is present, a wet compress may be placed upon the epigastric area and covered with gutta-percha tissue. A suitable abdominal binder of thin wool is often soothing to patients who go about, and is a protection to the sensitive abdomen.

Electricity is of doubtful value, but the proper regulation of rest and exercise (which latter should always be moderate) is of the greatest importance.

A change of scene, climate, and general surroundings is one of the most efficacious forms of treatment, both for its stimulating effect on the constitution generally, and more particularly as an aid in dispelling the accompanying neurasthenia. A sojourn at some of the numerous watering places is often of the greatest benefit, not so much on account of the waters taken (although they have their definite use), but on account of the diet and general regime under which patients are placed, and under conditions which are not usually followed at home. When this is impossible suitable exercise and properly regulated diversion are commendable, especially if

accompanied by the regular use of mineral waters selected according to the condition of the secretions.

Dietetic Treatment.—Dietetic treatment is of prime importance, although often hard to regulate because of individual preference. The diet must be administered with some regard to the severity of the case, but in all instances the food must be non-irritating and easily digested, and for this reason must require but little effort on the part of the gastric juice or muscular action of the organ. When only the scantiest diet is tolerated, milk, preferably diluted with lime-water, may be given at regular intervals, and this, while the patient is at rest, may suffice, provided he can be induced to take from one to two quarts per diem. We may say, however, that in the large majority of instances, a mixed diet, selected with reference to the needs of each case, is advisable.

When the motor power of the stomach is demonstrated to be normal, one may administer albumins, starches, and fats. If, on the other hand, it is deficient, it is wiser to exclude the fats. When, again, the secretory power of the stomach is defective, a mixed diet is very easily given, so long as the motor power remains unimpaired. In all cases, however, albuminous food should be finely divided. Carbohydrates should form the bulk of the food in those cases in which the hydrochloric acid is deficient, using preferably those in which there is least residue after digestion is complete.

Fats are imperative when malnutrition exists, and for these cases butter and cream form the most easily digested varieties. It is thus essential, where possible, to give a mixed diet, non-irritating, finely divided, and containing as much nutriment as possible within the smallest compass. In severe cases, in addition to the milk, one may give gruel, milk soups, light puddings, rice, arrowroot, toast, and then eggs. In some cases minced meats may be added; but it is not wise to add spices or any rich sauce. The craving which many patients with a chronic gastritis have for spicy things, under the impression that they will stimulate the functions of secretion, should not be encouraged. Of the lighter meats, etc., calves' brains sweetbreads, chicken, fish, minced beef, are those most preferable.

Alcohol is best avoided unless in the form of very light wine. The quantity of water taken with the meals should be restricted, unless there be hyperacidity, in which case it is well to dilute the food moderately. Instead of ordinary water, effervescing alkaline waters may be tried with benefit.

The determination of the proper number of meals per day is based upon the condition of the motor power. If this be good, three meals a day may suffice. Where, on the other hand, there is atony, four or five meals, each small in quantity, are more advisable. Weak coffee, tea, and cocoa may be given except in those cases in which hyperacidity exists. As the patient improves, such vegetables as spinach, carrots, maize, potatoes (mashed), and macaroni may be added in small quantities and gradually. Bread should be stale and not hot. When constipation is marked, it may be well to give stewed fruits, such as apples, prunes, etc.

Medicinal.—Inasmuch as hydrochloric acid is deficient in many cases, one may add with some benefit a few drops after each meal. Thirty drops of dilute hydrochloric acid in a glass of water, to be sipped at intervals for an hour after each meal, may be prescribed. On a scientific basis, there is little to be gained from the use of pepsin, although practically one frequently does find that its employment seems to afford considerable aid to

digestion. Pancreatin in doses of fifteen grains is perhaps better, and should be administered with soda, although rationally this should not be prescribed unless there is some evidence of atrophy of the gastric follicles. Papain is also recommended by many. After all, these artificially prepared ferments are realized to be of little use when one considers that the intestines carry on the defective actions of the stomach.

It seems of prime importance, however, to give some stomachic before each meal, and for this purpose one may try either dilute nitromuriatic acid, in doses of $\text{m}\times$ to xv (1 cc.), *nux vomica*, *quassia*, *gentian*, or *condurango*. In many cases the greatest relief from the pain incident to chronic gastritis is a pill consisting of silver nitrate, gr. $\frac{1}{6}$ (gm. 0.01); pulv. opii, gr. $\frac{1}{4}$ (gm. 0.016); extract of *hyoscyamus*, gr. $\frac{1}{2}$ (gm. 0.03).

When fermentation is an annoyance the diet should be carefully looked into; one may sometimes add thymol, carbolic acid, or creosote to the other modes of treatment. Germain Sée has recently recommended the use of strontium bromide, gr. 30 to 60 (gm. 2 to 4), especially when hyperacidity co-exists with the fermentation. For a distinct pyrosis, bismuth subnitrate and sodium bicarbonate, of each, 10 grains, combined with 3 to 5 grains of calcined magnesia, will usually afford relief. For persistent vomiting, lavage is the most rapid means of giving relief. When this is impracticable, a careful adjustment of the diet, with perhaps the administration of one or other of the usual drugs for that purpose, may be of benefit.

Constipation is one of the greatest annoyances. It is well that the patient should develop great regularity in his habits, going daily to stool at regular hours, whether there be need therefor or not. As soon as possible there should be added to his food vegetables containing much cellulose, also stewed fruits, especially a combination of figs and prunes; or in the early morning he should drink cold water or eat a fresh orange; and only in aggravated cases should we resort to either purgatives or enemata. Where purgation is necessary, it is a difficult matter to determine what drugs should be employed; only the mildest forms of purgatives should be given, and of these aloes and cascara are probably the most beneficial. The use of Carlsbad salts in the morning is another efficacious means of treatment in the aggravated constipation which often accompanies chronic gastritis.

GASTRECTASIS.

Definition.—Gastrectasis is an acute or chronic enlargement of the stomach cavity associated with a relative or absolute lack of power on the part of its muscular wall to propel the food to and through the pylorus within the normal time. In true gastrectasis there is permanent stagnation, *i. e.*, retention. It is a functional disturbance of motor power rather than mere enlargement of the organ.

Sometimes two stages are described: (1) Stagnation, with either simple or severe atony. The stomach retains some of the food between each meal, but not over night, and thus in the early morning it is empty and has a few hours of repose. (2) Retention, the extreme degree with no interval of gastric repose.

Introduction.—Modern methods of gastric analysis have demonstrated the importance and value of estimating the motor power of the stomach (*i. e.*, the ability of the gastric muscle to propel food into the duodenum).

In earlier days, when methods of examination were less accurate and less satisfactory, attention was directed more to the size of the stomach as an evidence of disease than to its physical and chemical functions. In this way extreme conditions only were observed, while the more insignificant varieties, in which the size of the stomach was often not much increased, were neglected. Increased capacity, however, does not in itself imply anything more than enlargement, and does not coincide with the idea of pathological function. The stomach is an organ whose size is subject to very wide variations in health, and clinical experience demonstrates the fact that mere increase in its dimensions does not imply a pathological state of the gastric functions. It is an every-day experience to find upon examination of patients that the stomach covers apparently and actually almost twice its normal area in the abdomen, and yet the subsequent examination with the stomach tube will reveal no disturbance of its motor function. Rosenbach¹ was among the first to point out clearly that such conditions cannot come into the category of "dilated stomachs" in the functional sense, and are certainly not included under the title of gastrectasis, which is less an anatomical than a physiological term. Some writers have employed the term *megalogastria* for the conditions in which there is mere enlargement of the viscus without any evidence of disease, and as such the term is useful not only in supposed congenital enlargement, but it may well be employed for the numerous instances of mere dilatation without impairment of motor power from whatever cause.

Probably the commonest form of gastric indigestion is that due to deficient muscular power—the so-called atonic dyspepsia—a condition of "slight motor insufficiency." Here again confusion seems to exist in the terminology. Atony of the stomach is after all but a milder form of gastrectasis, and while it may or may not be correct to place these two within the same category, the fact remains that in each the essential feature is a delayed expulsion of food from the stomach, through lack of power (relative or absolute) on the part of the gastric muscle. The term atony may be used also to imply mere laxity of gastric muscle without stasis of food, and such a condition is of course quite common. Certainly all grades of motor insufficiency occur from mere atony to extreme gastrectasis, although whether or not these can be etiologically linked together in one chain is not so certain.

Two conditions must exist to establish the rational use of the term gastrectasis. There must be an enlargement of the organ, and its motor power must be impaired. The deficient motor power may be merely relative, *i. e.*, the muscular activity may be even greater than is normal, and the fault may lie at the exit of the stomach. In other words, a pyloric stenosis may hinder the normally rapid outflow, and, even although the muscle be hypertonic, food may be retained. Such a gastrectasis would be relative.

As already mentioned, mere enlargement may be very marked, and yet, unless the motor power were disturbed, one would not be justified in regarding the condition as true gastrectasis. Vice versa, a stomach may be of normal size and its motor function so altered that food is retained for twenty-four hours or longer. Here, again, although the motor power is disturbed in the extreme, one cannot speak clinically or anatomically of gastrectasis. When,

¹ *Volkmann's Samml. klin. Vortr.*, 1878, No. 153.

however, the two conditions are combined, *i. e.*, increase in the capacity of the organ and definite diminution of its motor power, then, and then only are we justified in the employment of the term gastrectasis, to which, whether rightly or wrongly, a clinical entity has been ascribed. This terminology applies, however, chiefly to the chronic forms, for the acute variety of dilatation, which in most cases is associated either with pyloric stenosis or a sudden paresis of the gastric muscle, is in many respects essentially different from chronic dilatation and will therefore be considered separately.

Acute Dilatation of the Stomach.—This is probably more common than is generally supposed, and certainly as a terminal feature in pneumonia and cardiac disease, as well as a complication in surgical conditions, it is by no means rare. German students have been described by Riedel as commonly affected after beer-drinking bouts, and it has followed other errors and indiscretions in the diet. Brinton referred to the condition fifty years ago, although Fagge¹ first gave prominence to its clinical features. He recorded two fatal cases in patients eighteen and thirty years of age, respectively, in whom the stomach filled up rapidly with fluid. His description holds good to-day as one of the most complete pictures of the condition.

Campbell Thomson,² in 1902, had collected 44 cases, and Neck,³ in 1906, recorded 60 instances. The most recent reviews of the subject are that by Conner,⁴ who analyzed 102 cases of the disease (1907), and Laffer,⁵ who collected 217 cases in all. Doubtless many cases have not been recorded.

Etiology.—Various degrees of dilatation occur involving the stomach alone, the stomach with the duodenum, the stomach with the duodenum and jejunum, or the stomach with even a larger area of small intestine. This fact suffices to indicate that the causes must be varied and the pathogenesis different in various instances. There are predisposing and exciting causes, although it is not easy to dissociate these in all cases, all the more so as the etiology is apparently not only varied but often not to be determined at all.

1. *Debilitating Conditions.*—*General or Local.*—(a) *General, e. g.*, anæmia, rickets, toxic states, certain infections, especially pneumonia, typhoid fever and miliary tuberculosis, also cardiac disease (Goodhart⁶).

These may be the result of various influences which alter the muscular tone of the stomach wall, either indirectly by affecting nervous control, or more directly by toxic action on the muscle. There may be perhaps a neuroparesis, with spasm of the pylorus and stenosis, although, as Conner and others have suggested, the theory is unstable in view of the constant presence of bile in the vomitus in such cases. Nevertheless, the very sudden onset in many of these patients would suggest some special circumstance to which the toxic state or nerve lesion had led up. The condition is common in children with rachitic manifestations. In one case of pneumonia under personal observation the dilatation came on without warning, in a patient who had shown no signs of any gastric distress or abdominal distention half

¹ *Guy's Hospital Reports*, 1883, vol. xviii.

² *Acute Dilatation of the Stomach*, London, 1902.

³ *Centrbltt. f. d. Grenzgeb. d. Med. und Chirurg.*, 1905, Bd. viii, p. 529; also *Münch. med. Woch.*, 1906, Nr. 53.

⁴ *American Journal of the Medical Sciences*, 1907, vol. cxxxiii.

⁵ *Annals of Surgery*, March and April, 1908.

⁶ *Transactions of the Pathological Society of London*, 1883, vol. xxxiv, p. 88.

an hour before the extreme dilatation and agonizing symptoms had reached their full development. Lavage was at once performed, with immediate relief, and disappearance of the enormous distention tumor. Six hours later the symptoms recurred quite suddenly with similar results, the patient having rested quietly without signs of distress or distention in the interval.

(b) *Local*.—Previous gastric disease may predispose to the condition, usually through fermentation, giving rise to what has been termed the "gaseous form" of acute dilatation. This is often combined with the "fluid form." These terms, of course, represent symptomatic rather than etiological types. Excessive secretion has been regarded by some as the primary cause, although there is little to support the view.

2. *Trauma*.—Various forms of trauma are associated with acute dilatation: Head injuries (Erdmann, 1868, Thomson, etc.), with supposed lesions of motor nerves. Spinal injuries, with or without cord symptoms; in one instance (Edmunds) the dilatation appeared five weeks after a paraplegia. Blows on the abdomen (Wallace and Box).

3. *Postoperative or Postnarcotic*.—These cases, which are numerous, are remarkable in that few have been recorded in which operations on the stomach itself preceded the dilatation. Abdominal operations of various kinds (gall-bladder, uterus, ovary, kidney, appendix, etc.), as also operations on the extremities for a great variety of conditions have been followed by acute gastric dilatation. The interval between anaesthesia and the advent of gastric symptoms varied. In most cases the dilatation followed closely upon recovery from the effects of the operation and anaesthetic; in others, days or weeks intervened. James Bell's statistics from the Royal Victoria Hospital are of interest in this connection. There were 12 instances which followed various abdominal operations; some accompanied general peritonitis; in a few, definite gastromesenteric ileus was present, while in others no cause whatever was found. Several of the milder cases recovered completely.

The evidence tends to show that in many instances there is a parietic condition of the gastric muscle, associated with some toxic condition of unknown origin. Bloodgood¹ has recently given an analysis of cases observed by him after operation and in association with varying causes, one of the principal being the obstruction which occurs at the junction of the duodenum and jejunum (gastromesenteric ileus). In these cases the enormous distention of the stomach and duodenum is characteristic, and is to be differentiated from high occlusion of the small intestine. Doubtless with acute dilatation there may be additional dilatation of the duodenum with or without constriction at the mesentery, and in other cases, again, much of the small intestine may be concomitantly dilated. The diagnosis of the exact site of the obstruction (if this exist) has an important bearing on the selection of the treatment, both medical and surgical (*q. v.*).

4. *Traction on the duodenum* or pressure upon it from various conditions, especially a low situation of the small intestines in the pelvis, is no doubt a common cause. Here the tightened mesentery may incarcerate the duodenum as it lies on the vertebral column and produce the condition (Kundrat²). Albrecht,³ however, regarded this as a result, and not as the cause of the

¹ *Annals of Surgery*, 1907, xlv, p. 736.

² *Reviewed in Wien. med. Woch.*, 1891, No. 8.

³ *Virchow's Archiv*, 1899, Bd. clvi, p. 285.

dilatation. He believed that through dilatation of the stomach the small intestine was held low in the pelvis, and thus pulled upon the mesentery and indirectly compressed the duodenum. This "arteriomesenteric incarceration" thus aggravated the already existing gastrectasis. Whatever be the cause, certainly a large number of cases show at autopsy this constriction of the duodenum, and Bäumler's¹ beautiful anatomical proof is especially worthy of mention. In his classical case there was a very localized constriction near the lower end of the duodenum where it was crossed by the mesentery, and in the mucosa there was a well-defined circular necrosis corresponding to the external compression.

Albrecht had already demonstrated the fact that at this point the duodenum, even under normal conditions, is more flattened than round, because of the pressure of the overlying mesentery with its superior mesenteric artery, and, as Conner points out, this may be readily demonstrated in the cadaver, as also the increase in the constriction, by gentle downward traction on the mesentery with the finger inserted in the duodenum at this point. The three things considered essential to this are "the dorsal decubitus, an intestine nearly or quite empty of gas and feces, and a mesentery of suitable length—long enough to permit the intestines to slip into the pelvis and yet not so long that they may be supported by the pelvic floor." Perhaps it may be for this reason that lobar pneumonia is sometimes complicated by acute dilatation, owing to the weight of the lungs and violent coughing forcing the intestines down (Lehmann).

5. *Deformities of the spine* have been frequently observed in patients developing acute dilatation, and in many cases the orthopedic surgeons have been the first to observe the condition. Doubtless in these cases dietetic errors leading to fermentation and distention were often the exciting cause, while the restricted cavity of the trunk accounted in part for the onset of symptoms. In other cases perhaps the vagus has been indirectly acted upon and the control of peristalsis interfered with.

6. *Pressure of a kidney* which had been surgically fixed was the cause in one instance (Oppenheim).

7. *Dietetic Errors*.—Possibly too much or unsuitable food may directly induce acute dilatation by fermentative processes and distention; such are the repeatedly quoted cases where one meal or one indiscretion preceded the symptoms. Sometimes the combination of great fatigue followed by dietetic indiscretions has seemed to be the direct cause, and sometimes the compression of a plaster jacket, together with errors in feeding, may have accounted for the onset.

8. *Spasm of the cardia and pylorus combined* was believed by Kelling² to initiate the symptoms, although the frequent absence of these conditions scarcely bears out this view. Perhaps the association of hypersecretion with acute dilatation, to which special attention has been drawn by Heile, belongs to this category.

9. *The pancreas* is thought by some to have some relation to acute dilatation, and Bardenheuer³ mentions one instance following traumatic pancreatitis which was cured by gastro-enterostomy. Hoffmann, too, describes a case where chronic pancreatitis and multiple necroses were intimately associated with acute dilatation.

¹ *Münch. med. Woch.*, 1901, vol. xlviii, p. 357.

² *Arch. f. klin. Chir.*, 1901, 64.

³ *Deutsch. Chir. Congress*, 1904.

Morbid Anatomy.—The stomach is usually of enormous size, often extending down into the pelvis, and may be bent in an irregular U-shape, with the largest half at the fundus. The color is bluish, purple red, or pale; the wall is often thin. Definite pyloric obstruction is rare. The duodenum is dilated partially or throughout in half the cases. Sometimes the dilatation definitely stops near the lower end of the duodenum where the mesentery crosses, and may compress it against the vertebral column. At other times the dilatation extends throughout the whole of the small intestines.

Symptoms.—Mild and severe cases are found, the mild ones being slow of cure, with emaciation, weakness, and prolonged convalescence. The symptoms are usually acute and without previous signs of gastric disease, although closely resembling those of intestinal obstruction, for which, indeed, they are frequently mistaken. There is acute abdominal distress with distention of the area to the right of the midline and beyond it. There is pain in the epigastric or umbilical region, or perhaps in the left hypochondrium, with dyspnoea, increase in the pulse and respiration rates, but no fever. Vomiting, which is almost invariably present, appears early, as a rule, and is persistent. The vomitus is a dark-brown fluid, very abundant, often amounting to many quarts, containing bile and sometimes traces of blood. The odor is usually foul but not faecal. Analysis shows that HCl is a very variable quantity and that lactic acid is sometimes present. Hydrogen sulphide has been noted in several instances. Lavage, if carried out, empties the stomach of liters of such contents, the removal of which gives immediate relief. Constipation is the rule; the urine is diminished and of high specific gravity, because concentrated, and there is enormous thirst which is not easily satisfied.

Objective Signs.—*Inspection* often shows the outlines of a distended stomach, which disappear partially after vomiting, and totally after lavage. There is no visible peristalsis in the majority of cases. *Palpation* shows tenderness and sometimes the firm, tense cushion of the dilated stomach, and but little else, unless increased resistance in the left hypochondrium. Sometimes tenderness is absent, although this is not usually the case. A succussion splash is almost invariably obtained. *Percussion* shows diminution of the tympanitic gastric area and its replacement by dullness, especially in the left side of the abdomen and below the navel, reaching up as high as the right costal margin. This is an important diagnostic sign. In fatal cases there is collapse with the facies hippocratica; in cases with recovery the stomach returns to its normal size in a few days or weeks.

Diagnosis.—The condition is usually mistaken for some acute abdominal disease, especially perforative peritonitis or intestinal obstruction, and operation is frequently undertaken unnecessarily. The diagnosis requires a careful anamnesis. Of 60 recorded cases, only 13 were rightly diagnosed. The diagnostic features are the onset, with pain, collapse, and absence of fever, abdominal distention and localized left-sided dullness, with uncontrollable vomiting of large quantities of fluid containing bile, and having no faecal odor. The relief of symptoms and signs after lavage and postural treatment (knee and elbow position) are important confirmatory signs. The differential features of high intestinal occlusion are mentioned by Bloodgood as follows: "Initial pain, accompanied perhaps by peritoneal shock, which may later somewhat disappear, and vomiting without marked distention." In acute dilatation the initial pain is often absent, there is

epigastric distress and gradual and progressive collapse. Epigastric distress is early and relieved by lavage in acute dilatation, but is much later in high intestinal obstruction.

Prognosis.—The outlook is bad if the diagnosis be not made early and suitable treatment adopted; 47 out of 64 cases published died. In Conner's series of 102 cases, 74 died, although, as he remarks, many unreported cases recover where either the condition was mild or unrecognized. In 75 per cent. of Conner's cases the duration was less than five days, and in a few instances the patients died within three hours from the onset of the symptoms. If the complication does not end fatally, some impairment of motor power usually persists. The symptoms may disappear at once after treatment, or days and weeks may elapse before complete recovery.

Treatment.—Early lavage is essential, with the head low and the pelvis high; one is otherwise very apt not to reach the fluid at all. It is of great importance to thoroughly wash out the organ; at first often, later on less frequently. No food should be given by the mouth until recovery has sufficiently advanced, and one should use water enemata or subcutaneous saline infusions instead. Stimulants should be given as required. Operation is of no benefit unless after lavage there is no improvement, in which case some severe intestinal obstruction may be suspected. In these cases the probable incarceration of the duodenum requires special treatment. Schnitzler¹ first pointed out the benefits to be gained by turning the patients over from the dorsal position, and Bäumler recommended the knee and elbow posture as having been successful in two of his cases.

Where such methods fail, operation may be tried, although hitherto the results of gastro-enterostomy have been far from encouraging. Bloodgood's study of the condition has led him to believe that it is well to regard these cases as probable instances of gastroduodenal dilatation, and if lavage fails the duodenum should be carefully explored. If the dilatation be purely gastric, then gastrostomy or gastrojejunostomy should be performed, whereas if the duodenum too is involved, jejunostomy or duodeno-jejunostomy are preferable. Borchardt, quoted by Conner, states that where other methods of treatment have failed, it is doubtful if operation will meet with any greater success.

Chronic Dilatation.—Varieties.—There are two forms: (1) idiopathic, or absolute, otherwise called primary or atonic; simple gastrectasis; myasthenia gastrica; and (2) symptomatic, or relative, otherwise called secondary or obstructive—the result of stenosis of the pylorus—usually hypertonic. There is either benign or malignant obstruction. Rarely the duodenum is the seat of the stenosis. Allbutt suggests the term extension as including especially the cases of idiopathic dilatation.

A differentiation of the two main forms is not always possible, either clinically or even at autopsy, for cases with pyloric spasm, which has been the supposed cause of the atony, will often show no visible lesion. Such a condition would be in reality a symptomatic dilatation, and some authorities regard all so-called idiopathic dilatations as of this origin. The very existence of a true idiopathic condition is still a matter of doubt to some. While Stiller regards it as the most common form, Ewald and Fleiner admit that it may occur, and Strumpell and Boas regard it as a rare condition,

¹ *Wien. klin. Rundschau*, 1895, ix.

but admit that a mild gastrectasis may exist as the result of mere muscular insufficiency. Schreiber¹ and Ullman² entirely deny its existence, and the former states that certainly idiopathic dilatation, if it does occur, is never the result of simple atony. Crämer, too, is skeptical as to its existence, while Wegele³ regards it as rare and only associated with ptosis or with spasm of the pylorus. But if spasm were the main cause, hypertonus would certainly be more common than it is.

The diagnosis of idiopathic dilatation can only be made on the basis of prolonged observation to exclude stenosis. The proof of its existence lies mainly with clinical medical experience and surgical findings at operation. It has certainly fallen to the lot of every medical man of any experience to successfully treat individuals with a marked gastrectasis, and yet there is in these cases no development of signs suggesting a stenosis. In such instances gastric analysis shows retention of food, with all the evidence of gastrectasis, and the absence of further developments should suffice to prove the existence of a dilatation due to causes other than stenosis. Surgeons, too, find such a condition not uncommon, and operate for gastrectasis when careful search fails to reveal any signs of obstruction at the pylorus. Mikulicz, who has recorded such cases, evidently thought them not infrequent, and called attention to the fact that gastro-enterostomy in these patients often failed through the establishment of a vicious circle.

Idiopathic Dilatation.—The walls lose their resiliency and the viscus expands, becoming gradually more unable to handle the ingested food, especially when in large quantities. From various causes atony and temporary distention are produced. If the cause acts often enough, long enough, and severely enough, the elasticity diminishes, food is retained, and dilatation, subacute or chronic, is the result.

Etiology.—It occurs most commonly in adults of middle age, although it is not infrequent among rachitic children. Two classes of causes exist, local and general. It may be taken for granted that in many cases, simple atony, the result of manifold local and general conditions, is one of the most common predisposing antecedents of idiopathic dilatation.

Local conditions are trauma, improper clothing, laparotomy (Bennett's case followed an operation for umbilical hernia), repeated pregnancies, chronic gastric disease, especially chronic gastritis (with its favorable conditions for fermentation, which increases the distention of the weakened walls, and the excessive use of carbohydrate diet, which further tends to aggravate it). Dilatation may be the cause, accompaniment, or sequel of chronic gastritis; subacidity with fermentation and stagnation forms a vicious circle constituting Cohnheim's so-called vinegar and gas factory, and may likewise be regarded as an important etiological factor. Sometimes cancer acts in a similar manner without a co-existing stenosis, and also gastroptosis as part of a general myasthenia. Excessive work for the stomach from whatever cause, especially if the cause acts for a long period, is another predisposing antecedent; thus excess in quantity or quality of food, its mode of preparation (bulky, coarse, and indigestible), frequent and heavy meals with many vegetables, and much fluid frequently taken, *e. g.*, the forced milk cures, especially if combined with general illness, fever, etc., and

¹ *Arch. f. Verdauungsk.*, Bd. ii, 1896.

² *Münch. med. Woch.*, 1895, xv.

³ *Ibid.*, 1905, vol. lvii, p. 895.

little exercise, as happens so often with tuberculous patients in sanatoria. Beer is an important cause, although Allbutt has drawn attention to the frequency of dilatation in those who take excessively of temperance drinks, especially with much carbohydrate food. Excess of aerated water in any form is another cause. Hence, too, is the conclusion that gourmands may fare even better than the proletariat, because their diet is more varied and, therefore, their secretion is better.

The Relation of Motor Insufficiency and Gastric Secretion.—To a certain extent the propulsion of the gastric contents is governed by the amount and character of the secretion in the stomach itself (Pawlow). When the acid secretions are normal the amount of food passed through the pylorus seems to be controlled by the action of the duodenal mucosa. At all events only so much of the acid contents is passed as may be neutralized by the alkaline juices from the pancreas and accordingly the pylorus is closed or left open; when no acid exists the propulsion is rapid, for neutralization is not necessary. On the other hand, with hyperchlorhydria there may be stagnation of food (and certainly this often does exist), although at present it cannot be definitely stated that the one is the cause of the other. Again, hyperchlorhydria and hypersecretion frequently exist in the absence of dilatation. It is doubtless true that hypersecretion may cause pyloric spasm and indirectly induce dilatation, all the more so if the retained food be rich in carbohydrates, which may induce fermentation and thus add to the causes of gastrectasis. The importance of atony in the symptomatology of pathological secretion of HCl in gastric disturbances is shown by Kaufmann. His views regarding the condition of the secretions and their relation to the causation of symptoms are briefly as follows: He has drawn attention to the fact that in patients with variable degrees of HCl secretion the symptoms depend on the motor power rather than on any disturbance of secretion per se. Many patients with typical symptoms of hyperacidity show normal acid values on analysis, and, vice versa, many individuals in whom the symptoms of hyperacidity disappear do not show on analysis a synchronous decrease in the acidity. He therefore concludes that in hyperacidity some other condition must complicate matters to produce the symptoms thereof, and this factor is the atony. The same applies to cases of subacidity and achylia; one finds either no symptoms at all, and in these instances the motor power is good, or else if symptoms accompany such gastric findings, then atony must be present.

He thus describes two classes of patients: (1) In the one there is variable secretion (high or low), with atony; the symptoms are really due to the atony, and are cured by lessening it. (2) In the second there is variable secretion without atony. Such cases come under the category of neuroses, *e. g.*, hyperæsthesia of the gastric mucous membrane, and resemble similar conditions in other organs (*e. g.*, cardiac neuroses, nervous diarrhoea without excessive mucus, dyspnoea without lung trouble, etc.); *i. e.*, gastric neuroses so-called. Such patients need no local therapy for the stomach but rather general treatment. In such cases the secretions remain abnormally high or low, and yet the symptoms disappear because depending on a general neurosis, which when cured is accompanied by an alleviation of the local symptoms.

Certainly, in gastrectasis the HCl secretory changes alone are mere accidents in the symptomatology and must not be given too prominent a place.

Usually when the motor power is repaired any symptoms supposedly due to hypersecretion or hyposecretion will disappear.

Relation of Movable Kidney to Gastrectasis.—Up until quite recently opinions varied widely on this topic, and ever since Bartels first noted the possible relationship, writers have dealt with the theories of cause and effect between these two conditions. Litten found movable kidney in 55 of his cases of gastrectasis, while, on the other hand, Lenhartz did not find movable kidney in 16 instances of gastrectasis, and denied any relationship.

Certainly, one has ample evidence that movable kidney is extremely common in women, and yet gastrectasis is not by any means frequent. Certainly, too, in these cases there is often some atony of the stomach, but rather as a part of a general enteroptosis or general asthenia. Again, when gastrectasis occurs it is more than probable that one kidney at least (and usually the right) is more or less movable—but in such instances there is frequently some degree of gastropptosis as well—and the downward dislocation of the two viscera is attributable to a third and common cause.

Relation of Gastrectasis and Gastropptosis.—It may be stated in general terms that gastrectasis (*i. e.*, the extreme form of atony with enlargement of the stomach and marked motor insufficiency) is nearly always accompanied by a certain amount of gastropptosis, and the lesser curvature is found lower in the abdomen than is normal. It may further be said that gastropptosis is much more common than gastrectasis and that where it exists as a primary condition it does not usually bring on gastrectasis of an extreme grade, for in the majority of cases of gastropptosis the motor power is found upon analysis to be fairly good. Schreiber describes two forms of gastrectasis: (*a*) the descended form, following a previous gastropptosis, and (*b*) the undescended form, in which the stomach is prevented by adhesions or some other cause from altering its position.

It is quite conceivable and it very often happens that when gastropptosis exists in excessive eaters the persistent overloading of the stomach will cause more and more descent of the organ, with the result of finally causing a kink at the duodenum—as may occur in the cases which show the crescentic horizontal variety of gastropptosis. A progressive stenosis with increasing motor insufficiency may occur. Steele and Francine found on examination of 70 patients with gastropptosis that dilatation existed in all. The degree, however, must be very slight when one considers how extremely common it is to find gastropptosis when absolutely no signs or symptoms of indigestion exist. This fact alone should indicate that except under certain other predisposing conditions, gastropptosis does not generally cause gastrectasis, even though it frequently precedes and more often follows it.

General Causes.—An hereditary weak stomach is recognized as a factor of some importance, and predisposition to chronic dyspepsia certainly exists as a family weakness. Chronic constipation is regarded by Ewald as a source of gastrectasis. The persistent inactivity of the intestines or their paresis leads to diminished gastric peristalsis and atony. So also may any intestinal disease.

General causes assist the local conditions and thus aid directly and indirectly in inducing dilatation, for local causes act more forcibly in stomachs rendered susceptible through prolonged illness or general myasthenia, be it organic or neurotic. The cause may be toxic or muscular, *e. g.*, in sepsis,

typhoid fever, tuberculosis, and pernicious anæmia (whether it be that toxins weaken the wall or the muscle be altered through the anæmia and malnutrition. The same holds in chlorosis and in repeated hemorrhages). Toxic causes act either generally, *e. g.*, tobacco, or both generally and locally, as in the case of alcohol. A busy life with no rest after meals and hasty eating is often an antecedent cause. Thus it is with musicians of sedentary habits, neurotics, and hypochondriacs. Venereal excesses are regarded by some as a factor in the etiology. Anything which induces general weakness may be regarded as a predisposing general cause.

Relative, Symptomatic, or Obstructive Gastrectasis (from Stenosis).—The causes of stenosis are numerous: spasm with gastric ulcer, erosions and scars (never from neuroses) (Cohnheim). Foreign bodies: *e. g.*, resin balls from swallowing shellac spirit for ten years, hair balls, cherry stones (Crämer); gallstones and beans and peas (Fleiner); such bodies may remain for three months. Hypertrophic pyloric stenosis; polypi; tumors (Crämer); ulcers with scar formation causing puckering at the orifice or tumor formation; cancer of the pylorus and of the lesser curvature extending to the pylorus.

Tumors outside of the stomach (*e. g.*, of the liver or gall-bladder, pancreatic tumors pressing on the duodenum, floating spleen with pressure on the duodenum, movable kidney, dermoids, enlarged glands, etc.) may be a cause.

Perigastritis: inflammations outside of the stomach and associated with neighboring organs, etc., *e. g.*, liver, gall-bladder, pancreas. Cicatricial puckering associated with cholelithiasis, perigastritis, etc. Epigastric hernia. Appendicitis with or without adhesions. Omental adhesions from appendicitis, especially if to the linea alba. Torsion, flexure, and kinking of the duodenum at the duodenojejunal fold in enteroptosis. Trauma, producing inflammations near the pylorus, hæmatoma of the wall, or spasm of the pylorus. (The theory of spastic stenosis of the pylorus with temporary insufflation implies ultimately weakness of the wall.)

Pathological Anatomy.—The dilatation varies in degree and character. The one anatomical feature is the altered size of the organ, although that is not always increased even with marked motor insufficiency; sometimes it is monstrous and may seem to fill the entire abdominal cavity. In Strauss' case of stenosis from an old ulcer the stomach held five and a half liters, and one hand could be placed between the lesser curvature and the liver, and yet there were adhesions to the gall-bladder. The intestines were above and the stomach below. Much larger dilatations are mentioned. Jadow's case in which the capacity of the organ was 45 liters is worthy of mention, if true.

The gastrectasis may be partial and is sometimes diverticular. Dilatation begins in the lowest portions and those most overtasked, the fundus, pylorus, and greater curvature. Later it becomes general. The other abdominal viscera are in extreme cases compressed and pushed in various directions. The spleen and liver lie abnormally high, as a rule, while the small intestines are pushed downward.

The thickness of the stomach wall varies. In one case with chronic inflammation (Lebert) it was 14 mm. thick. It is especially in the obstructive cases that this increase occurs, although even in idiopathic cases it may be abnormally thick. As a rule, when no stenosis exists the gastric wall is

thin and atrophic. The mucous membrane may be thickened, hypertrophic, or irregular and hard, a condition usually existing when chronic gastritis is also present. Sometimes the mucosa is thinned and atrophic, and not at all infrequently atrophy and hypertrophy alternate in the same mucous membrane.

The muscle is changed, its appearance depending on the cause and pathological condition. With pyloric stenosis, especially if benign, there is muscular hypertrophy (compensatory), while in idiopathic cases the atrophied muscle is but a feature of the general myasthenia. Fatty and colloid changes are often seen in microscopic preparations. The interstitial tissue is often infiltrated in the chronic cases with increased fibrous tissue and small round cells, while in the acute cases there may be small round cells, extravasated blood cells, and separation of muscle and fibrous tissue cells.

Symptoms.—The general appearance of the patient varies according to the cause. With idiopathic dilatation and benign stenosis there is usually some emaciation, but a fair color is preserved. If malignant disease be present, there is usually some evidence of cachexia. The skin is naturally dry because of retention of fluids in the stomach. There may be urticaria and erythema. The extremities are often cold.

Dyspeptic symptoms develop early. The appetite is usually diminished (unless hyperacidity exists from some benign stenosis), the breath is sour, and flatulence and pyrosis are present. A sensation of fulness is an early symptom, and yet lavage may show a normal motor power at this stage. Thirst is an important and often a distressing symptom. Water is not absorbed in the stomach, so that in gastrectasis, if the water be retained and is not passed on into the duodenum, it is practically outside of the body. The degree of thirst corresponds to the amount of retention and depends on the degree of motor insufficiency, of which it is therefore a good criterion. Vomiting merely increases the thirst.

Constipation is marked, because no water is passed into the intestines, and there is atony of the bowel. Dry, hard scybala are often passed with colicky pains; piles and fissures form. The degree of constipation is also a good criterion as to the degree of motor insufficiency. *Diarrhœa* is rare, sometimes mucous colitis co-exists, and sometimes diarrhœa and constipation alternate.

Emaciation is usually marked in advanced cases because no nutriment is absorbed. It occurs in proportion to the degree of dilatation, especially with diminished secretions, and is worse if the intestines are deranged.

Pain is less marked in idiopathic than in obstructive cases, and is in part due to the weight of food in the overloaded stomach. No severe pain exists in idiopathic cases. In obstructive cases, especially if from ulcer or spasm or cancer, it is often intense. Sometimes, even in cancerous dilatation, no pain exists. When present it often radiates to the breast or back.

Vomiting is not so constant in idiopathic as in obstructive cases. In the latter it is the rule because the food cannot pass, and the stomach empties itself by emesis. A feature of the vomiting is its periodical occurrence, with ever-increasing short intervals. It usually occurs after every few meals only, and is explosive. The amount has no relation to the previous meal, and the vomitus consists of food from days or weeks before. It is thus copious, and may amount to several quarts. Later there may be little

or no vomiting because the walls are weakened, a condition which is of course serious. The general appearance of the individual foodstuffs depends on the character of the gastric secretions. The color is usually of a dark gray brown. There are three layers, froth on top, fluid in the middle, while the sediment below contains yeast, sarcinae, mucus, and food (new and old). (Allbutt denies this and says sarcinae and yeast are rare.) Mucus, if abundant, indicates a probable gastritis. Microscopically, one sees bacteria, fatty acid crystals, epithelial cells, sometimes blood cells and pigment. There is an odor of fermentation; organic acids may be abundant. The finding of lactic acid is important only when in the fasting contents or after a test meal; moreover, Strauss¹ maintains that lactic acid from ferment action does not appear in the stomach if 0.12 per cent. HCl (total) is present. Vomiting gives temporary relief even although it only partly empties the stomach. With the vomiting the appetite improves and the urine increases in quantity. HCl may be in excess, deficient, or normal.

The Urine.—Corresponding to the amount of retention, so is the diminution in the renal secretion. It forms a good basis for measurement of the gastrectasis. Some classify cases of gastrectasis into three grades, estimating the degrees according to the quantities of urine, as 1000 to 1500 cc. for the mildest, 500 to 900 cc. for the medium, and under 500 cc. for the most severe grades. The quantity of urine is also a guide to prognosis and treatment. Small amounts indicate a more serious form, and suggest the use of water enemata at regular intervals. Increase in the urine indicates improvement. The urine when diminished is concentrated and has a high specific gravity. The chlorides are lessened and the phosphates increased. If much hyperacidity is present the urine may be alkaline. Albumin and also acetone and diacetic acid are sometimes present.

Cardiovascular symptoms are common, especially palpitation, sometimes even anginoid attacks. There is frequently marked throbbing of the abdominal aorta. Secondary anemia is often marked. The pulse is often feeble, slow, and irregular. Gastrectasis is often the last straw in old heart lesions, infections, etc. Temporary dyspnoea may occur, but true asthma is rare.

The *temperature* is lowered. Patients with gastrectasis usually feel chilly from slight causes. More rarely there are febrile attacks from a concomitant gastritis or toxic causes. Weakness is usually present and the patients tire easily.

The nervous symptoms in gastrectasis are varied. Languor, apathy, depression of spirits, and melancholia are common features. Headache and dizziness are frequent. "*Vertigo a stomacho laeso*" of Trousseau has long been recognized. It differs from auditory vertigo in being less sudden, and rather a subjective unsteadiness, than a rotatory or other displacement of objects. Mild psychoses, insomnia, deafness, etc., are common manifestations, and when coming on at a regular time are regarded by patients as the cause rather than the effect of the malady. These nervous phenomena are probably the result of dryness of the tissues, inanition, and auto-intoxication.

Tetany is worthy of special notice. Mentioned by Morgagni, it was first described by Newman in 1861, and later by Steinheim, while Kussmaul gave it prominence as a classical feature of dilatation in his description

¹ *Zeitsch. f. klin. Med.*, 1895, Bd. xxviii, p. 567.

of gastrectasis and its treatment by the stomach pump in 1869. This condition, although common to various diseases of the gastro-intestinal tract, is chiefly associated with pyloric obstruction in which dilatation, hypertrophy of the stomach wall, with hyperacidity and hypersecretion are present. It is thus more commonly associated with benign stenosis, and is rare in obstruction from malignant disease or from external pressure. It is probably induced by the absorption of some soluble poison which is unneutralized in the stomach. The attacks appear after severe and persistent vomiting, and after lavage, and are associated less with the act of vomiting than with the presence of putrid contents in an overfilled stomach. The stomach should be emptied some hours before lavage is practised to avoid the onset and recurrence of the attacks. The duration may be from a few minutes to hours, days, or weeks. When prolonged it ends usually fatally. Mild grades are common and probably often overlooked. Muscular twitchings, prickling sensations, etc., in gastrectasis are doubtless warnings to urge surgical intervention. Cr  mer's view was that any gastrectasis not vastly improved in four weeks under lavage, dieting, and massage should be subjected to operation.

Various theories exist as to the causation of tetany. Kussmaul regarded it as due to dehydration of the tissues—nerves and muscles—on the analogy of the occurrence of cramps in Asiatic cholera, an analogy which, however, is scarcely justifiable, inasmuch as the same cramps occur in the so-called cholera sicca in which dehydration of tissues takes no part. Later on Kussmaul himself disclaimed the theory, although Fleiner, Albu, and others still maintained it.

Germain S  e held the theory of its causation by reflex action from stimulation of the sensory nerves of the stomach, *e. g.*, after lavage, vomiting, etc., and its frequency in sucklings, its occurrence with intestinal parasites and disappearance after their removal, suggests this possibility. Gerhardt regarded the complication as the result of a poison, basing his views on the nervous symptoms, some of which resembled those of alkaloidal poisonings. Bouveret and Devic¹ originated the theory of auto-intoxication from the prolonged action of abnormal processes of digestion, and this theory holds its pre  minence up to the present time. In proof thereof they isolated a substance soluble in alcohol, resembling Brieger's peptotoxin, which when injected into animals caused general convulsions. Opponents of these experiments held that the convulsions were not of the nature of tetany, that alcohol alone could induce similar attacks, and that untreated gastric contents are incapable of inducing such symptoms. Dickson tried the injection of gastric contents in various ways to produce tetany, but without success.

Indican has often been found in the urine of patients suffering from gastrectasis with tetany. Acetone has been found in the stomach, but not in enough quantity to do harm, because oxidized.

The prognosis in tetany is always grave, and the average mortality has been given as 70 to 80 per cent. There were 31 deaths in 40 cases (Albu). Death usually follows only after the tetany has been well developed, from failure in treatment. Patients who pass successfully some attacks of tetany, are liable to recurrence of attacks.

¹ *Revue de M  d.*, 1892, p. 38.

Treatment of Tetany.—Medical treatment may be tried for a short time, but surgical interference should not be delayed too long when lavage and medicines fail. Careful washing of the stomach should be beneficial. Greenfield suggested after careful lavage with some mild antiseptic a final washing with a solution of sodium phosphate and then the introduction of about ten ounces of boiled milk containing sodium phosphate in the proportion of one dram to a half pint, diluted with hot water. The one efficacious method, however, is the surgical one which provides proper drainage by performance of a gastro-enterostomy.

Physical Signs of Gastrectasis.—These vary greatly, especially according to the cause and extent of the condition, for cases of primary or spastic dilatation show no tumor.

Inspection.—This is very important. The prominence of the stomach may be visible, although in mild cases there is usually nothing abnormal seen. The stomach is seen better if stenosis exists, because it is then less flabby and more hypertonic. The abdomen is usually flabby, the stomach much enlarged, and the outline of the greater curvature may perhaps be visible down as far as the pubis. This is most easily seen in women. Unless ptosis is also present the lesser curvature remains high in the epigastrium. The position of the lesser curvature alone determines the presence or absence of gastropptosis. In uncomplicated cases, usually the bulk of the organ is above or about the umbilicus and perhaps to the left, in contradistinction to mere ptosis when it is below. Although usually big, it need not be larger than normal. It may seem intermittently dilated (as Bennett described), by some movable obstruction, *e. g.*, kidney, pediculated papilloma, etc. In fat people one sees little on inspection, but these patients usually become thin sooner or later, and then inspection often reveals the diagnosis.

Visible peristalsis is rare without obstruction. With pyloric stenosis it is either spontaneous or induced by flicking the abdomen with a wet towel or the finger. A tumor-like prominence appears in the left hypochondrium, which swells, falls, and re-appears to the right. Antiperistalsis sometimes exists.

Inflation is best performed by the use of Davidson's double bulb syringe attached to the stomach tube; less satisfactorily by administration of tartaric acid and sodium bicarbonate, because the dosage cannot be gauged. Before employing this method, one must be careful to exclude ulcer or fresh adhesions, and should never distend the organ to an extreme degree.

Palpation should be done systematically, carefully and gently with the flat hand first, then following the respirations, examining with the patient in various positions, and even in doubtful cases with the aid of an anæsthetic if necessary. In idiopathic dilatation one gets merely a sense of the air cushion present, and this more definitely tense with obstruction, while sometimes one can feel gas passing the pylorus. Boas lays stress on the importance of palpating for contraction of the stomach, both stiffening and rigidity (so-called *Magensteifung*), and peristalsis.

The *percussion splash* has a certain value. The presence of a superficial splashing sound, however, probably implies merely a lax condition either of the abdominal muscles or even of the stomach muscle itself, but does not prove the presence of motor insufficiency. Deep splashing, which is elicited in the early morning, or seven hours after food has been taken, is strong evi-

dence of gastrectasis. If with this one can obtain a succussion splash as well, the diagnosis is rendered still more certain. Splashing sounds on palpation or percussion over the large intestine are sometimes hard to distinguish from those produced in the stomach. In the former case diarrhoea or other intestinal symptoms are present, and the same splashing may be obtained in the other portions of the colon far away from the stomach. The splashing which neurasthenics can voluntarily induce by contraction of the abdominal muscles is of course not to be confused with the above, for it may be performed without gastrectasis and even when very little fluid is present.

Percussion determines the size of the organ, or at all events its outlines. There is a deep tympany over the stomach, the extent depending upon its size and situation. Dehio's percussion tests after drinking water may be mentioned. Dulness may be found below when the patient is in the upright posture, if much fluid is present. Sometimes the dulness is more to the left, and changes with position and after emptying the stomach. This is merely a slight aid to diagnosis and only to be used with insufflation tests as well.

The liver dulness is usually absent, especially if adhesions tie the stomach to the diaphragm. Absence of liver dulness depends, too, on the position of the hepatic flexure and is not in itself a reliable sign of any pathological state. Percussion affords us no information as to function.

Auscultatory percussion has not as yet given the satisfaction hoped for it; Broadbent recommends it, Allbutt does not. Certainly control tests by various methods do not prove it infallible for outlining the borders of hollow viscera. Too much depends on the nearness of the scratching or percussing finger and on many other factors which alter the note.

Examination with the *x*-rays after the administration of bismuth subnitrate (30 gm. in a thin soup) may enable one to see the outlines and the less active fundus; one may also see the pylorus open intermittently and the impaction of masses in imperfect solution prolonging the closure of the pylorus. But nothing directly of value for the diagnosis of dilatation is afforded by means of the *x*-rays.

The salol and potassium iodide tests are unreliable. The desmoid test is also of doubtful value, depending much on the quality of the material used.

Stomach Contents.—The essential feature is the abnormally large quantity of food returned after a test meal. This alone is a proof of motor insufficiency, and is pathognomonic. Chemical examinations give varying results according to the primary cause of the dilatation. In the milder forms, depending on idiopathic conditions, HCl is usually diminished or normal, and rarely increased. In the obstructive forms it is excessive or normal, rarely diminished unless late on, and then chiefly in cancerous and atrophic conditions. Bile may or may not be present. Varying evidences of fermentation exist (lactic acid, yeast, gas, etc.), according to the conditions present. Thus, while lactic acid is commonest in malignant disease, gaseous fermentation with hydrogen sulphide is usually an indication of benign stenosis. The production of gases is not prevented by the presence of HCl, nor is albuminous putrefaction; in fact, the latter is usually only present when HCl is there. The motor insufficiency, not the secretions, is the *cause* of the gas fermentation. The state of the secretions determines the degree

of digestion of foodstuffs after test meals. Microscopically the findings differ according to the cause of the dilatation, *i. e.*, whether benign, malignant, etc.

Diagnosis and Differential Diagnosis.—The various considerations may be thus tabulated:

1. As to the presence of gastrectasis.
2. If present, as to its nature, *i. e.*, idiopathic or obstructive.
3. If obstructive, as to the nature, *i. e.*, benign or malignant stenosis.
4. If probably benign, as to its special form, *viz.*, spasm, foreign body, hypertrophic stenosis, intragastric tumor, extragastric tumor, inflammation, etc.

The presence of gastrectasis is determined chiefly by the symptoms and signs of advanced motor insufficiency. Very often the *symptoms alone* make the diagnosis obvious. The vomiting of large quantities of food retained beyond the usual time is in itself an indication of motor insufficiency, and the amount is moreover to some extent a guide to the degree in which this exists. So also is the degree of thirst and the amount of urine passed.

If, however, the symptoms be less marked, the use of the *stomach tube* can alone determine with accuracy the presence of the gastrectasis, for the mere proof by insufflation of a large stomach does not exclude the condition of megalogastria, which may be present without any sign of motor insufficiency. Nor should we rely too much on the *succussion splash*. When the stomach is full, it may be quite absent, and if no vomiting has occurred the observer may be quite deceived. Its presence is not always synonymous with gastrectasis. Certainly opinions vary greatly as to its diagnostic value. While Stiller regards its presence long after the ingestion of food as pathognomonic, Elsner and Kuttner look upon it as merely an evidence of lax musculature with or without gastrectasis.

It is best to begin with an examination in the early morning after a full meal on the previous evening. If, under these conditions one finds no food remnants and not even sarcinæ, gastrectasis certainly does not exist in any advanced degree. If food be present (and, normally, there should be no remnants at all), then we have motor insufficiency. We usually get very little fluid in fasting contents, less than 40 cc. If solid parts remain, it suggests a probable obstructive dilatation, and the more solid, the worse that condition is. Even if only a little mucus and sarcinæ be found, we have some abnormal condition, *viz.*, either early insufficiency or a healing process. Sometimes we get sarcinæ in the fæces too. Thus if such a patient takes much unsuitable food, the healing condition will retrogress and once more show insufficiency.

It is essential to *make a microscopic examination always*, first of the fasting contents for food. If this be present, no further test is needed. If no food is seen, then one should examine microscopically for sarcinæ and yeast. The presence of lactic acid is also important, for any of these are suspicious signs of motor insufficiency. Fleiner says rightly that every ectatic stomach has motor insufficiency, but not every stomach with motor insufficiency is necessarily ectatic, *e. g.*, cirrhotic stomach. Usually the motor insufficiency is the prelude to the gastrectasis.

Winkler and Stein¹ suggest the use of iodipin, an iodine compound in oil. This sets free iodine in the intestine only, and if present in the saliva it indicates that it has passed the pylorus, an evidence of a fairly good motor power.

Gastrectasis must be distinguished from several conditions.

From Dilated Colon.—One must make repeated examinations at various hours for signs, and empty the stomach by siphonage. It is often hard to tell without such tests, and it may be well to inflate the colon from the rectum. The history and symptoms will assist in the decision. Absence of splenic dulness in itself is of little importance in the diagnosis, being merely an aid. One must exclude emphysema, pneumothorax, and diaphragmatic hernia. Moreover, one must look for a cause, movable or diseased kidney, umbilical hernia, tumor, etc. If necessary, an anæsthetic should be used.

From Gastropptosis.—In simple dilatation, the lesser curvature is in its normal position, while if there be gastropptosis, the site will be lower than normal, and the position of the stomach may be vertical, horizontal, or otherwise. In simple gastropptosis there is no sign of motor insufficiency after a test meal.

From Nervous Dyspepsia (without Motor Insufficiency).—The symptoms are out of all proportion to the organic changes, and there are various stigmata of neuroses.

From Chronic Gastritis.—The stomach is of normal size and the motor power normal or nearly so (unless dilatation co-exists, as it usually does later on). There is a history of the cause, and much mucus is vomited.

From Hypersecretion.—After complete lavage of the stomach on the previous evening there is abundant gastric juice found in the early morning (more than 40 cc. should be present to make a diagnosis of hypersecretion).

From Gastric Crises.—These present one feature resembling gastrectasis, viz., the marked and copious vomiting, not, however, of retained food, but of concentrated gastric juice and mucus. Gastric digestion is usually normal in the intervals between the attacks. Other conditions, *e. g.*, ovarian tumors, dilated gall-bladder, sacculated peritoneal exudates, and pancreatic cysts should offer no difficulty.

Sometimes a *spindle-shaped dilatation of the œsophagus* may occasion doubt because of the periodical vomiting of food from previous days. This may be hard to differentiate. The stomach, however, is not enlarged, the introduced tube causes immediate vomiting of alkaline material, while if the tube be pushed farther it reaches the stomach and withdraws acid contents.

The Differentiation Between Idiopathic (Atonic) Gastrectasis and Obstructive (Hypertonic) Gastrectasis (from Pyloric Stenosis).—Advanced cases are usually readily diagnosed but in early stages of the disease, and even in some that are of long standing, the differential diagnosis may be impossible for a long time. As a rule, one must take the whole history, symptom complex, and signs into consideration. The following table indicates in a general way the essential features:

¹ *Centrblt. f. inner. Med.*, 1899, No. 33.

	Idiopathic dilatation.	Dilatation from pyloric stenosis.
History.	None, except of dietetic excesses.	Previous ulcer, hemorrhage, gallstones, carcinoma, etc.
Course.	Slow.	Rapid.
Emaciation.	Slow.	Rapid.
Pain.	Little or none.	Marked.
Vomiting.	Infrequent and copious, liquid and incomplete. Less painful.	Frequent, copious, thick, and mixed with much solids, and complete. Accompanied by pain.
Dilatation.	Moderate as a rule.	Extreme.
Visible peristalsis.	Absent.	Present, and often very marked or easily induced.
Tumor.	Absent.	Often present.
Lavage.	Quick inflow, slower egress.	Perhaps slow ingress, but rapid exit. Expression is easier and often explosive.
Therapeutic tests.	Improvement marked with lavage, etc.	No improvement, but progressive decline in spite of lavage.

Benign pyloric stenosis gives special features to the clinical picture of gastrectasis. The earliest physical signs arouse suspicion when the past history yields some cause for a stenosis. Distress appears soon after dietetic errors and the symptoms increase slowly but progressively. The two predominant subjective features are pain and vomiting. The two predominant physical signs are visible peristalsis and evidence of a tumor. Gastric analysis usually shows increased hydrochloric acid secretion, and microscopically there are yeast cells and sarcinae.

The pain soon follows upon the early feelings of mere distress. It comes on chiefly after food, and, while periodic or intermittent, it recurs with ever-increasing frequency and often with progressive intensity from week to week, and sometimes is almost continuous. Later on, as retention of food occurs, the pain may be nocturnal too, associated perhaps with hyperacid secretion, fermentation, or dependent on the special nature of the obstruction at or about the pylorus. Often the pain is definitely associated with the increased peristalsis, and is therefore sometimes of a cramp-like nature, especially if hypertrophy of the gastric muscle has occurred. The vomitus, which in the course of the malady follows closely upon the onset of the pain, may consist chiefly of solids. Vomiting occurs often with increasing frequency up to the time when hypertonus gives way to atony, and then the emesis recurs at longer intervals and is less explosive in character.

Physical Signs.—The early stages may reveal nothing but a prominent epigastrium or a slight fulness, but visible peristalsis soon appears and should be carefully looked for. Often much patient watching is required before the peristalsis appears. Inspection, further, often reveals the pyloric tumor, sometimes as movable, shifting with respiration or by alterations in the position of the internal viscera. Palpation reveals increased resistance over the pyloric area, and a cushion-like sensation over the viscus itself. In thin, flabby persons the tumor may be palpable quite early. Movability is a characteristic feature of non-adherent pyloric tumors resulting from scarred ulcers. They may be often shifted to widely different areas of the abdomen, are usually not very sensitive, and do not seem to be very nodular.

Bouveret has described a sign which he calls intermittent gastric tension. In the recumbent posture the left side of the epigastrium is more prominent than the right, and on gentle palpation is balloon-like to the feel; during palpation, however, the swelling disappears, re-appearing very soon, off and on, and especially after the ingestion of food.

Varieties of Pyloric Stenosis.—*Pylorospasm.*—This occurs at the height of digestion, as a rule, and may be constant or periodical, resulting in temporary closure of the pylorus with retention of food. There may be severe pains, vomiting, and signs of motor insufficiency, such as thirst, diminished urine, constipation, and enlarged gastric area. The vomitus is hyperacid, and there is often hypersecretion. The vomiting relieves until the cycle recurs. Analysis of the contents shows stasis, increase of HCl, sarcinæ, yeast, fatty acid crystals, and signs of marked fermentation. The pyloric area is tender and may show tonic contractions, while palpation of the fundus may reveal the characteristic rigidity (*Magensteifung*). The prognosis is more favorable, although recurrences are frequent.

Cicatricial Stenosis of the Pylorus (the type is the scarred ulcer, although caustics, etc., may induce it).—The history is of importance, especially the record of previous hæmatemesis or of recurrent attacks of vomiting and pain ("indigestion"). The signs of progressive stenosis, as seen on physical examination, gastric analysis and examination of the stools for occult bleedings, are likewise important.

Duodenal stenosis may be indistinguishable, unless the obstruction be infra-ampullary, in which case there will be a constant back flow of bile into the stomach. Stenosis of the duodenum usually occurs from cancer, pressure of benign tumors, scarred ulcer, localized peritonitis, gallstones, malformations, etc.

Perigastric Adhesions with Pyloric Stenosis (cholelithiasis, inflammations, etc.).—Here, too, the history is of great importance, also the physical signs, the presence of jaundice previously, and the existence of traces of bile in the urine.

Stenosis from external pressure occurs from enlarged glands, dermoids, new-growths, and inflammatory exudates, also from kinking or constriction through fibrous bands, etc., as also from traction as occurs in acute dilatation where the duodenum is constricted through traction of an arterio-mesenteric fold.

Impaction of food or foreign bodies in the pylorus may be the cause or benign tumors within the pylorus, *e. g.*, polypi.

Hypertrophic stenosis, the so-called stenosing gastritis. The condition is probably a rare one, arising from different causes and presenting as its two main features at first a chronic gastritis and later the signs of motor insufficiency with stenosis. There are no cicatrices and no tumors, but a diffuse thickening of muscle or fibrous tissue may be present, a localized benign stenosis.

Quite a few isolated cases are on record (Boas, Habershon, Tassia, Tilger, etc.). The condition has been found most commonly in middle age, runs a slow course over years, with exacerbations and remission of symptoms (chiefly those of gastritis followed by stenosis). There is anorexia and emaciation. The gastric analyses vary. It is not easy to distinguish from a scarred ulcer which may have been latent for years, but the diagnostic features are those found in the general picture with its protracted duration.

In recent descriptions by Russell and by Maylard,¹ cases of adult stenosis of congenital origin are described with symptoms of varying intensity, and these writers conclude, whether or not on sufficient proof, that such cases are very common indeed. It seems, however, not always easy to prove the existence of a stenosis during operation, and the mere improvement after gastro-enterostomy should not of itself convince one that a pyloric stenosis had been present—still less that it was congenital.

Stenosis from Malignant Disease of the Stomach.—The following table will outline the essential differences from benign conditions causing stenosis:

	Benign.	Malignant.
History.	Ulcer, toxic gastritis, caustics, etc.	Previous normal digestion, perhaps.
Age.	Any age.	Usually after forty years of age; not infrequently earlier, however.
Course.	Rapid, but with less marked decline in health.	Rapid and progressive development of weakness, etc.
Tumor.	Smooth, often more movable.	Nodular, hard, fixed.
Secretions and contents.	HCl normal or excessive; no lactic or butyric acid; H ₂ S present sometimes; few or no Boas-Oppler bacilli; sarcinae long present.	HCl less or absent; lactic and butyric acids often present; H ₂ S uncommon: Boas-Oppler bacilli common; sarcinae uncommon, and soon disappear.
Therapeutic tests.	Sometimes nutrient enemata benefit.	Nutrient enemata useless.
Metastases.	None.	Usually sooner or later present.

Course and Duration of Gastrectasis.—Acute cases have already been referred to, and their duration varies from a few hours to several weeks, this depending largely on the nature of the cause; mild, and severe cases occur, and most of the severe types end fatally in a short time. Chronic gradual dilatation is the more common condition, and the course and duration vary according to the etiology. The cause determines not only the duration but also the essential features and symptoms of the malady, giving to the clinical picture perhaps great suffering in one instance, while in another there may be comparative comfort.

Idiopathic cases are slow in development, and their course depends on the persistence of the exciting cause and the possibility of its removal. Patients who have long been the victims of the disease in an advanced stage will be difficult to relieve. With early and suitable treatment the condition very often becomes permanently held in check, and development will extend over years. Thus the idiopathic cases and those due to pylorospasm, being more amenable to therapy, will run a mild course and last a shorter time than those associated with stenosis. Pyloric obstruction causes a more or less rapid development of symptoms, rapid where the obstruction develops quickly, as in carcinoma, subacute perigastritis, etc., more slowly with gradual cicatrization of an ulcer at the pylorus. Where benign stenosis exists, its special nature, its degree, extent, and curability, the condition of the muscular wall of the stomach, and the early diagnosis determine the probable duration. If not relieved by appropriate treatment these patients get rapidly worse and fail from inanition; surgical intervention (gastro-

¹ *British Medical Journal*, 1908, ii, p. 68 and 71.

enterostomy), on the other hand, may alter the whole course of the malady, and quickly bring about recovery even after advanced disease exists and after years of suffering.

The stenosis of luetic origin may improve under specific treatment, or else the newly formed scars which result from the effect of specific treatment upon the healing process may only increase the stenosis. Cancerous stenoses, of course, run a progressively downward and usually a rapid course unless removal of the neoplasm is carried out. Under rare conditions, however, they improve markedly for a time, either because the pylorus is reopened through breaking down of the obstructing cancerous mass, or because of the effects of general treatment.

The concurrence of gastritis and intestinal disease lengthens the duration of the malady and adds to the difficulties of cure. Relapses are very common in the idiopathic cases, and are usually dependent on indiscretions of diet or excesses in daily routine. Persistence in the exacting treatment is by no means easy, and the tendency to relax in care makes it difficult for patients to lead a model life of the kind required for permanent cure.

Prognosis.—In the early stages of gastrectasis one should carefully guard against a too definite assurance as to the cause and probable outcome of the affection. The two features which govern the prognosis are the cause and the possibility of its removal. Cures are possible in idiopathic cases that are even advanced, provided the wall of the stomach is not permanently damaged and the general condition of the patient capable of improvement. Gastropnoia renders these cases much more difficult of cure. Early cases are of course always amenable to cure if the proper treatment be employed and conscientiously carried out. The normal amount of urine, the lessened degree of thirst, the good condition of the musculature, and possibility of obtaining suitable diet are all favorable features. The onset of tetany is a serious complication and one of grave omen. Relapses are common and due recognition of their frequency is important in dealing with questions of prognosis.

In obstructive cases the cure is always possible, except in malignant disease, for the possibility of satisfactory surgical intervention, when a new opening may be made to short-circuit the passage, renders a benign stenosis of no importance in itself.

Treatment.—A detailed diagnosis as to the nature of the gastrectasis is essential to rational *treatment*.

Prophylaxis.—Proper hygiene, diet, and mode of life are essential to the prevention of atony of the stomach, as is also moderation in the use of food and drink. Special care in debilitated states, as regards diet, exercise, and habits, must also be carefully remembered, and one should avoid the previously mentioned causes of idiopathic dilatation.

The treatment should be based on (a) the etiology as regards food, drink, and habits, and (b) the social state, history, family history, previous diseases, and conditions of the viscera.

In all patients with motor insufficiency certain general rules apply, no matter what the cause or underlying condition may be. The stomach must be as far as possible emptied within the normal time, and stagnation and retention of food avoided. Food must be administered of a kind which, as regards quantity and quality, will not excessively burden the stomach. The motor power being weak, one must aid its activity by avoid-

ing unnecessary effort on the part of the gastric muscle, while fermentation must be avoided or reduced to a minimum.

The Diet.—The main objects to be obtained are as follows: (*a*) To supply sufficient nutriment in an easily assimilable form, (*b*) to avoid overburdening the weakened stomach with bulky foods, (*c*) to avoid fermentation and retention of foods, (*d*) to give foods of such kinds and in such a way as to assist their early propulsion into the intestines where the chief digestive function is carried on, and (*e*) to supply enough fluid to the body to fulfil the requirements of tissue thirst.

These results are best obtained by giving frequent small meals, six small meals daily, rather than three of ordinary amount. Food must be given either in soupy form, or as dry, solid food in a finely divided form. It must be taken slowly and well masticated; for this reason the teeth must be kept in good condition. The patients must rest before and after food, preferably in a recumbent position, on the right side. Occasional lavage assists materially. This principle depends on the evidence given some years ago by v. Mering that water is not absorbed in the stomach, so that its propulsion into the intestine is necessary for its usefulness to the tissues.

In any case much fluid should be avoided, no matter how great may be its nutritive value, for gastrectasis has been well called a dyspepsia of fluids.

Whatever of fluids be taken they should be sipped slowly. Milk, being one of the most perfect foods, is also one of the most suitable in gastrectasis. It is well to add some cereal to it, *e. g.*, barley, rice, oatmeal, etc., or one of the numerous concentrated albuminous foods. Or one may add cream, or make a custard. Milk is often distasteful to patients who suffer from retention of food, and for this reason any ordinary flavoring extract may be added to aid in its administration. The addition of lime-water or brandy may be desired and may effect the same purpose. Bouillon with eggs makes an excellent form of concentrated nourishment, and is usually readily assimilated; so also are meat juices and meat jellies. The dry foods and those more solid should be prepared in a finely divided form, especially the meats and vegetables.

Whatever meats are used should be chopped up and well masticated. Scraped raw beef, tender roast beef or fillet, thinly sliced smoked ham, oysters, and sweetbreads are often well borne, as also chicken, pigeon, tender mutton, and white fish of various kinds. Vegetables must be mashed or in purée form. Only the less coarse varieties and those with least cellulose should be employed. Purées of potatoes, peas, spinach, asparagus tops, or cauliflower can usually be taken with ease. Bread may be given, preferably white stale bread, or toasted, or one may use rusks and unsweetened biscuits. Fats, notably milk fat, are good, especially with the addition of HCl. Alcohol is of value only when weakness is great and small quantities of stimulant are necessary. It should be remembered that although absorption occurs in the stomach, the alcohol at the same time induces an increased secretion of water into the stomach. Malt extract in small amounts may aid the appetite and general strength.

To some extent the diet must depend on the results of gastric analysis. With *subacidity*, cereals are preferred rather than albumins, and whatever meats are used should be finely divided before eating. White meats are preferable, and dry white fish is usually well borne. Vegetables, even though

better taken than proteids, must be finely divided and given in limited amount. In these cases the use of drinking water to which a little diluted HCl has been added is often beneficial; it should be sipped slowly after food. With *hyperacidity* one should select mainly albuminous foods, meat, milk, eggs, and fats in moderate amount, of which butter is the most easily borne. Starches should be carefully restricted, although rusks and dry toast are suitable. Sweets should either be much limited or altogether avoided. Fluids are more tolerated than in subacidity, especially the still alkaline waters. *Saline enemata* are often most useful to relieve the tissue thirst in idiopathic dilatation.

Mechanical Measures.—The most important is thorough lavage of the stomach. This is indispensable in the medical treatment of motor insufficiency if that condition be at all marked. In the mild cases it may not be required, although it often affords great relief. It is not merely a form of symptomatic treatment, but is even curative in the early cases. As a rule, however, early mild cases merely require strict attention to diet and daily regimen to effect a cure. One must judge according to the individual case how much lavage is necessary. In the mild forms of motor insufficiency where stagnation occurs only after a heavy meal, there may be no occasion for its use, although often an occasional artificial emptying of the stomach gives prolonged relief. This is best done in the late evening.

In the moderately severe cases the stomach is never empty during the course of the day, food being retained until the subsequent meal, but the stomach empties itself during the night. In such cases a careful lavage given before the evening meal does good by removing the fermenting products of the day's feeding, and a small appropriate meal taken thereafter on an empty and cleaned stomach has a good opportunity for absorption with the patient at rest for the succeeding ten hours.

In the very severe cases when food remains in the stomach overnight, and has not been propelled into the intestine by early morning, lavage is still more imperative, and in the most extreme forms it may be well to wash the stomach twice daily, in the early morning as well as in the evening. The essential is that the lavage be thorough, and that the return flow of water be finally quite clear. It may be necessary to wash out the stomach with the patient in various positions, standing and lying down. Much time and patience may be needed, and often an hour of lavage is required before the water returns perfectly clear. Lavage should not be left indiscriminately to the patient, but the physician does well in most cases to perform it in person or by a trained assistant. Lukewarm water suffices for the purpose, although when much fermentation is present, salicylic acid solution (1 to 1000) may be used. How long should one keep up lavage? This must depend on the persistence of the motor insufficiency. It should be done daily until we are sure that digestion is not delayed overnight. The lavage should be discontinued gradually, daily treatment giving place to one washing on alternate days, the condition of the contents deciding the subsequent frequency.

Other mechanical measures are of less importance, although often of benefit. Massage is beneficial in a general way, but should be used systematically, and under proper skilled direction. In extreme cases of gastrectasis it is not well to employ vigorous massage to the abdomen, for it is not supposed to benefit the muscle of the stomach directly, but assists more by increasing the general muscle tone, by improving the circulation, and aiding

the nervous system. It also assists in obviating the chronic and often most persistent constipation. It should not be done until at least one and a half hours after any meal. When much fermentation exists, or where there are organic causes for the dilatation, it is well to avoid the upper abdomen and confine the treatment to the lower zone and to the general musculature elsewhere.

Hydrotherapy is of the greatest benefit; in fact, apart from lavage, there is no mechanical treatment so beneficial in gastrectasis. Warm shower douches gradually made colder, with the use either of the strong hose stream playing up and down the back, the use of the Scotch douche, the employment of salt rubs, or of douche massage give eminently satisfactory results. Where hydropathic apparatus is not available, much benefit is gained from daily cold baths, followed by dry friction with a rough towel. The use of compresses over the stomach at night covered with oiled silk and an appropriate binder often affords great ease to the patient.

Bandages of a supporting nature, or proper-fitting corsets for women which support from the pelvis, are useful; such corsets are constricted about the hips, but are loose about the epigastrium and lower part of the thorax, thus affording room for breathing, and not allowing the stomach to be without support from below. These are especially useful where gastroptosis exists.

Electricity has merely a general value in the same way as has massage. Its direct local action is very dubious. Experiments on dogs have shown that faradism does not directly affect the gastric muscle, but causes contraction merely of the external abdominal muscles. Apart from this general effect, electricity, even if applied as intragastric faradism, is but another form of suggestive therapeutics. The same applies with even more truth to the use, or rather the abuse, of static electricity and high-frequency currents.

Medicinal.—Drugs form the least satisfactory of all methods in the treatment of gastrectasis. No drug that is known can effectually contract the dilated organ or act with continuous benefit upon the gastric muscle. Strychnine is much used for this purpose, although but little evidence exists to prove that its value is other than as a general tonic. The drug therapy is purely a symptomatic one. When hypersecretion exists, relief is often obtained from atropine in small doses ($\frac{1}{100}$ gr.) at night, or if need be in the morning as well. Alkalies have perhaps an indirect value in neutralizing the already existing excess of acid, although, of course, they have no effect in diminishing the secretion. For subacidity the use of well-diluted HCl sipped in a tumbler slowly every few minutes after food seems to aid digestion, or at all events to give added comfort. The use of bitter vegetable tonics is much lauded.

In moderately severe cases the writer has repeatedly seen beneficial results from the use of olive oil, in dessertspoonful doses after food, as was recommended by Cohnheim. This author, however, preferred its administration through the tube in doses of 200 to 500 cc.

Antifermentatives can only be of use in the milder cases, but with marked prolonged retention their use can have but little effect. Probably salicylic acid is one of the most satisfactory.

Constipation requires varying treatment according to its degree of severity. In mild cases no drugs are required, and proper feeding, massage, exercise, and perhaps occasional water or soapsuds enemata will alone suffice. It is

often useful to administer the Rochelle or Sprudel salts in small doses in the early morning, after which the patient takes moderate exercise for a short time previous to breakfast.

In any case drastic purgatives do harm, and only mildly acting drugs are to be recommended. Should these fail in the more severe forms of dilatation, reliance must be placed on enemata every other day. Daily evacuations are not necessary to health, and patients should be advised accordingly.

Vomiting should be controlled by proper diet and by lavage. When these are ineffectual, nutrient enemata should be administered, and in persistent cases it will be necessary to call for surgical intervention. For the thirst the proper use of saline rectal injections will aid in relieving the distress, and the mouth should be frequently rinsed with some pleasant wash.

Surgical.—The indications for operative interference include three types of cases:

1. Acute cases when medical treatment has failed or is known to be useless, *e. g.*, with twisting of the duodenum.

2. Pyloric stenosis—whether for removal of adhesions, fibrous bands, tumors obstructing the orifice, external compression from any cause, or when relief is impossible unless one is enabled to create a new passage for the food.

3. Idiopathic dilatation without marked gastropnoia or general enteropnoia, when medical treatment has been unsuccessfully tried, and where, in consequence, the patient is losing in weight and strength from insufficient nutriment, or again, when the symptoms, even though not grave, are sufficiently irksome to interfere with daily routine.

Two objects are to be attained: (1) To remove the cause. (2) To remove the results. The methods¹ are: (1) Resection of the pylorus, pylorotomy. (2) Heineke's pyloroplasty. (3) Gastro-enterostomy. (4) Bircher's gastroplication, which reefs up the anterior wall and diminishes the size of the stomach. This latter was advocated by Keen, Weir,² and Brandt.³ Eight of Bircher's ten cases did well. Gastro-enterostomy in uncomplicated cases has given excellent results. Enteropnoia is distinctly a contra-indication to surgical interference—and even mere gastropnoia, if at all marked, is not likely to permit of successful operation in cases of gastrectasis, even although the gastrohepatic omentum be shortened to aid in removing this complication.

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

Nomenclature.—Congenital pylorospasm, hyperæmesis lactentium (M. Schmidt), infantile hypertrophic stenosis of pylorus. The views as to its origin render the nomenclature involved. Pylorospasm and systolic stomach are different conditions from true hypertrophic stenosis of the pylorus.

Historical.—Osler mentions Hezekiah Beardsley as being the first to record a case (1788). The patient died at five years of age, having had

¹ See articles on gastric ulcer and gastric cancer—surgical considerations.

² *New York Medical Journal*, July, 1892.

³ *Centrblitt. f. Chir.*, 1894, No. 16.

the symptoms from infancy. Later, Williamson, in 1841, and Daworsky in 1842, mention cases. Landerer¹ (1879) and R. Maier² (1885) described anatomical findings in patients, from twelve to eighty years of age, which they regarded as congenital, and Hirschsprung³ (1887) described congenital pyloric stenosis in two infants. There are now more than 150 cases recorded. John Thomson's publication on the subject in 1897 did much to draw the attention of the medical profession to its frequency and importance.

Morbid Anatomy.—Two distinct conditions seem to exist (perhaps a third with mild hypertrophy and an abnormally small lumen).

1. **Pylorospasm.**—Pylorospasm without true hypertrophy is described by Pfaunder⁴ in systolic and semisystolic stomachs, a pseudohypertrophy. He describes three states of the pylorus at postmortem. (a) The diastolic, in which the muscles are relaxed and relatively large, the normal condition. (b) The systolic, in which the muscles are contracted. (c) The semisystolic when the pylorus is contracted and the rest of the stomach is relaxed. Pylorospasm includes mere spasm, readily overcome by any relaxation of the sphincter, and seen at operation and postmortem as a contracted pylorus. Fixation occurs at death in one phase of digestion. Heubner and Freund⁵ share this view of the underlying cause. It is possible that some of these cases, which show symptoms during life and are recovered from, are due to temporary blockage from a mucous plug or from a swollen membrane, as a result of gastric catarrh. These cases must be regarded in the light of mild types, not requiring surgical intervention because there is no anatomical disturbances of a permanent nature. In a sense they are pure neuroses.

2. **True Congenital Hypertrophy.**—The pyloric ring is increased in size and thickness, owing to hypertrophy of the muscular fibers, chiefly the circular ones, although it is difficult to measure to what degree the longitudinal muscle shares in the increase. Its color is somewhat paler than the surrounding structures. The ring is from 1.5 to 3.5 cm. thick, forming a cylindrical swelling, often very firm, sometimes fusiform in shape, according to the degree of hypertrophy. The fusiform shape is due to traction of fibers on the duodenal end. The swelling is sharply delineated at its duodenal end, but diminishes gradually toward the fundus. The mucous membrane is folded up, either in several folds, or sometimes at one place only, thus resembling a single tumor, likened to the verumontanum of the prostatic urethra. Such a case was described by Arreger⁶ as one of tumor in a child five years old, although, from our present knowledge one may conclude it to have been a case of pyloric stenosis. The submucosa is little, if at all, altered. There is in most cases a marked absence of the products of inflammation, although some cases record fibroid thickening as well. The lumen is small. The stomach is dilated, and in cases of long standing often shows a diffuse hyperplasia of its muscle. The duodenum is empty.

A third type may be mentioned in which there is chiefly spasm, but with a mild hypertrophy which does not seem to increase and which is amenable

¹ *Congen. Hypert. Stenosis of Pyl.*, Tübingen, 1879.

² *Virchow's Archiv*, 1855, cii.

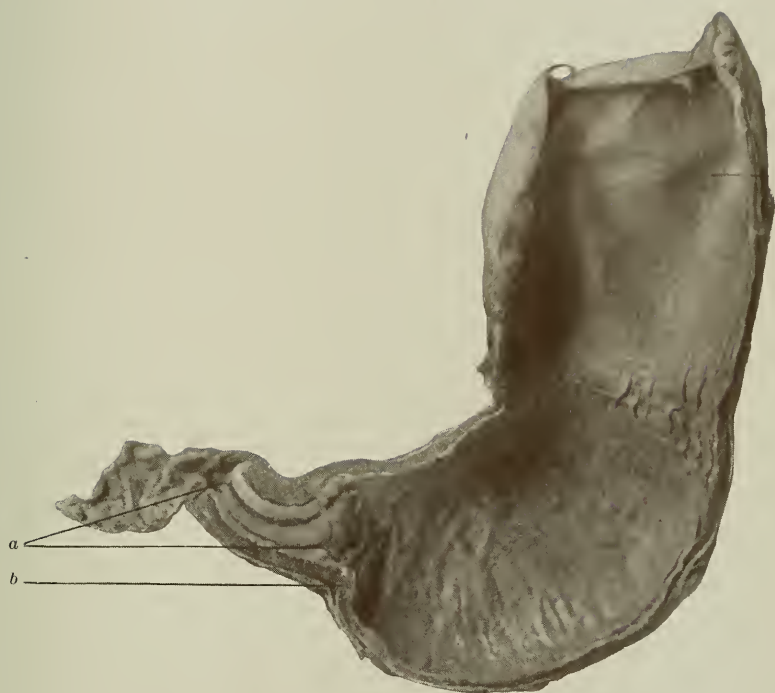
³ *Jahrb. f. Kinderheilk*, 1888, vol. xxviii.

⁴ *Wien. klin. Woch.*, 1898, No. 45.

⁵ *Mitt. a. d. Grenz. d. Med. u. Chir.*, 1903.

⁶ *Ueber Pylorusstenose in Kindesalter*, Inaug. Diss., Zurich, 1896.

PLATE IX



Congenital Stenosis of the Pylorus.

a, pylorus; *b*, hypertrophied muscularis.

to medical treatment alone. Such cases have been demonstrated in the autopsy room.

Pathogenesis.—The recent work of Cunningham,¹ Stiles,² and others has thrown much light on the anatomical condition. Normally, the stomach may be considered as an organ in which the pylorus is a distinct and separate portion, with a function both to expel and to retain food. The cylindrical canal, which is about 2 to 3 cm. long, is contracted at birth, and its lumen is obliterated by the folds of mucous membrane. The ring projects normally into the duodenum in a way similar to that in which the cervix protrudes into the vagina. The muscular fibers of the pylorus are so arranged that the circular ones, which surround the whole length of the canal, are to some degree antagonized by the longitudinal bundles, which as Cunningham shows, are derived from the body of the stomach, and spread about, in gradually increasing numbers over the pylorus, some going to the duodenum, but most of them stopping short at the pylorus, and dipping even into the submucosa, aiding in relaxation of the sphincter. Between the circular fibers of the pylorus and those of the duodenum there is a dividing band of connective tissue. In the cadaver the pyloric canal is usually tightly closed, so also probably during life except during digestion, when it opens irregularly and intermittently. When pyloric stenosis exists, the whole canal is probably involved and the changes are primary, while any lesions in the stomach elsewhere are secondary.

Various theories are held. Those congenital cases in which strictures are found due to foetal peritonitis, syphilis, neoplasms, etc., do not come into this category. Regarding the condition in the light of a true hypertrophy, one necessarily excludes theories of functional pyloric spasm ("systolic stomach"), as suggested by Pfaundler. Opposed to Pfaundler's views is the fact that systolic stomachs at postmortem are not so large and rarely as firm as those which have been described as true hypertrophic stenosis. Moreover, the diastolic stomach, as described by Pfaundler, should be of wider caliber than the contracted one, and yet no proof of this exists. Nevertheless, doubtless many cases both at operation and at autopsy are wrongly regarded as true hypertrophic stenosis, when mere systolic contraction of the pylorus is present.

Other conditions of a mild nature, such as recover readily on medical treatment, may be regarded as due, in most instances, to obstruction of the pyloric lumen from swollen membrane or an inspissated mucous plug, a condition, too, that one might readily conceive to be associated with spasm. Such a case was described by Scholten, in which a true thickened membrane was observed at operation and at postmortem. On opening the abdomen there was partial relaxation and gas passed through the pyloric ring. Spasm, too, is often seen in laparotomies, in the pure hypertrophic cases, even in spite of the use of chloroform. Again a mild muscular hypertrophy may exist when the symptoms are amenable to proper treatment and recovery occurs. Some, at least, of Heubner's and Koplik's³ cases may have been of this nature.

¹ *Transactions of the Royal Society of Edinburgh*, 1906, vol. xlv.

² *British Medical Journal*, 1906, ii, p. 943.

³ *American Journal of the Medical Sciences*, July, 1908,

Doubtless the earlier observers had the correct view in regarding the condition as developmental hyperplasia of the pyloric muscle, a congenital redundancy, or prenatal overgrowth (Nicoll,¹ Cautley,² Ibrahim³). The fact that the pylorus first appears in foetal life at the third month shows that enough time exists before birth for excessive growth, while the degree often found excludes the possibility of postnatal development. Sometimes where pyloric stenosis occurs as a congenital condition, other parts of the body show abnormalities; *e. g.*, there may be congenital atresia of the anus, and perhaps, as Stiles suggests, we have an analogous condition in incontinence of urine with overdistention of the bladder, overcoming finally the sphincter vesicae. Other analogous conditions are pathological dilatation of the colon, stenosis of the sigmoid, and dilatation and hypertrophy of the rectum.

Torkel,⁴ in 1905, cites an instance in which, on morbid anatomical grounds, a proof exists of a congenital origin in that aberrant embryonic tissue is found amid the fully developed structures. He suggests that there is a hyperplasia of the wall of the pylorus which according to the degree of development may induce more or less stenosis. In most cases described there has been a diffuse hyperplasia, which shows, however, merely the normal formation of muscles as seen in the gastro-intestinal tract. But in this hyperplastic tissue one may get inclusions (*Einschlüsse*) which must be regarded as aberrant germ cells and which show that we are probably dealing with developmental disturbances. Nor is it necessary that such inclusions be present in all cases, but the fact of their being present in some shows the tendency and the relation between developmental disturbance and hyperplasia in general. The hyperplasia may be the sole evidence of developmental disturbance. In Torkel's case there happened to be aberrant Brunner's glands.

A nervous theory was advanced by Thomson and Still, according to which the condition was associated with disturbed coördination of the relaxing and contracting fibers of the pylorus, with increased irritability, and this latter, whether originating during intra-uterine or extra-uterine life leads to spasm, and this spasm leads to hypertrophy, an hypertrophy from overwork. In many specimens examined there is hypertrophy of the circular muscles chiefly and to a less extent of the longitudinal fibers, suggesting that the latter were overworked and hypertrophied in an effort to antagonize the sphincter action of the spasmodically contracted circular bundle—in other words an antagonistic dilating force was induced to overcome the closure by the circular sphincter, and thus hypertrophy of both bundles occurs. However, no proof exists of incoördination or of nervous derangement in foetal life. Nor are we sure that spasm causes the hypertrophy of the pylorus. If this were the case, the same spasms might be expected then from other conditions of irritability, *e. g.*, hyperacidity and faulty feeding, especially with artificial foods, but such facts have not been shown to occur. Freund, however, regarded hyperchlorhydria as an important etiological factor. Henschell's view, according to Stein, is that it exists from a family tendency to hypertrophies, but this is not proven. Finkelstein⁵ thinks the cases are mostly acquired as a result of proliferative inflammation causing secondary hypertrophy.

¹ *British Medical Journal*, 1904, ii.

² *Transactions of the Royal Medico-Chirurgical Society*, 1899, lxxxii.

³ *Munch. med. Woch.*, 1905, vol. lii, p. 674.

⁴ *Virchow's Archiv*, 1905, vol. clxxx.

⁵ *Jahrb. f. Kinderheilk*, 1896, xliii.

Symptoms.—The condition is more frequent than was supposed until recently. The infant, frequently the first born and as a coincidence more often of the male sex, is healthy at birth, and shows symptoms at any time from a few hours or days after birth up to two or three months of age, but not later. The average time of onset is from the third to the sixth week, and very rarely as late as the fourth month. Very few cases are on record in which the symptoms began immediately after birth—a point of great importance in the diagnosis. Ladd has such a case on record with successful surgical treatment. Signs of digestive derangement are the first to appear. Early anorexia, often overlooked, may usher in the symptoms. The breast is taken reluctantly and the child is easily satisfied. When vomiting begins early, however, the appetite is often maintained in a striking way. Vomiting is the chief early sign. It occurs whether the child be breast or artificially fed, and no matter how carefully this may be done. At first it seems unimportant, although no adequate cause is found; then its persistence rather than its frequency attracts the attention. It may not be frequent at first, occurring once or twice daily, or perhaps after each meal if the child be well fed, or only after an interval of several hours.

When careful small feedings are given, or if the food be weak, fluid and non-coagulable, the vomiting is less frequent, but the quantities are larger than is the amount of the meal last given. Lavage, too, will naturally cause vomiting to diminish in frequency and amount. The vomiting is forcible in character—an explosive ejection to some distance, expelled perhaps through the nostrils, as in older children. Where much retention exists the amount is copious, and the contents vary in character. HCl and lactic acid may or may not be present; there is mucus, and sometimes blood, fresh or coffee-ground in appearance, but no bile. Other features are the absence of nausea and any permanent effect from diet, relief after vomiting, and the maintenance of a good appetite, amounting even to hunger.

Signs.—With the vomiting one finds a clean tongue and sweet breath. Constipation is the rule, requiring the use of enemata, or more rarely diarrhoea with loose, green, watery stools containing mucus, and perhaps tarry from the presence of blood. The urine is diminished. The temperature is irregular because of the toxæmia, the body is cold, the pulse small, weak, and quick. Wasting is sometimes rapid, and there may be marked weakness, the result of the starvation, the condition resembling and often being mistaken for marasmus. The child is often in severe pain, with violent cramps and epigastric resistance, and cries out or whines; at other times it is quiet and dull. The abdomen is sunken; the intestines are undistended. These symptoms may remit for a while, but usually recur after a short interval.

The Dilatation.—Sometimes the stomach region bulges if the organ be full, and the dilated viscus may be seen extending to the navel. Dilatation is often marked; in one of Henschell's¹ cases the stomach held 400 cc. The presence of dilatation depends upon the duration and degree of obstruction; hence, it is more marked in the late stages. Heubner found the majority of cases with undistended stomach, which he attributed to the constant vomiting. Dilatation is perhaps more common with bottle-fed infants.

The visible peristalsis is most marked in long-standing cases, although Sarvonat and Audry² described it in a child aged two weeks. Probably

¹ *Archiv f. Kinderheilk.*, 1891, xiii, p. 32.

² *Lyon Méd.*, 1905, civ, p. 1013.

when mere pyloric spasm exists, without true hypertrophy, there is much less peristalsis. It is an important sign when present, although not always found, and often only at long intervals, especially after food. One must, therefore, wait to observe it, and it may be necessary to induce it by flicking with the finger, a cold towel, etc., or to watch while feeding. The waves are usually very obvious when present. This sign should not be confused with the muscular action of the recti nor with abnormal peristalsis of the transverse colon when turned upon itself. Antiperistalsis is rarely found.

The Tumor.—Palpation (which should be made with warm hands) is important, especially in the region just outside the right nipple line and one-third of the distance from the umbilical level to the costal margin (or half an inch to the right and three-quarters of an inch above the umbilicus). Sometimes lower than, and sometimes beneath the enlarged liver, one feels a hard cylindrical or spherical lump, the size of a filbert, which may even be felt to contract and to change in hardness during peristalsis.

The Diagnosis of Congenital Hypertrophic Stenosis (not Pylorospasm).—The symptom complex gives the clue. (1) The time of onset and the age; symptoms develop in early infancy, but not immediately after birth. (2) The vomiting, which is persistent, forcible, occurs always after meals, but also regardless of feeding, in healthy children. The vomitus does not contain bile. (3) The absence of milky stools. (4) The visible peristalsis. (5) The tumor, which is palpable as a finger-like mass, and movable downward. It is not always to be felt, as it sometimes lies under the liver, in which case one must diagnose by exclusion and by a careful and rational experimenting with food. (6) The dilated stomach. (7) The crying from hunger and pain. (8) The emaciation.

Differential Diagnosis.—Congenital hypertrophic stenosis is to be distinguished from: (1) Simple regurgitation in which the above symptom complex is absent. (2) Stenosis of the duodenum; kinking from shortened ligaments, adhesions, etc. (3) Tumors near the duodenum, such as enlarged glands narrowing the lumen by pressure from without. (4) Congenital atresia of the pylorus, which is a rare condition due to developmental error and rapidly fatal. It is therefore incompatible with life, unless relieved by operation. Vomiting occurs immediately after birth, even if food is withheld, or in other cases as soon as this is given. (5) Malformations and atresia of the small intestines. There is bile in the vomitus and the duration is short, at most a week. Sometimes an atresia occurs at the pylorus or in the duodenum, which is incomplete at first, so that the failure of health is more gradual, and the stomach may be hypertrophied in consequence. (6) Gastritis. (7) Simple vomiting and constipation from faulty feeding. (8) Habit vomiting. (9) Pylorospasm due to faulty feeding. Here the signs and symptoms are similar, but milder, as a rule; there is less visible peristalsis and the contracted nodule is usually smaller than the tumor of true stenosis. The condition, too, is more amenable to treatment.

Prognosis.—This depends to a large extent upon one's ideas as to what is meant by the term pyloric stenosis. Wherever the symptoms are due to pylorospasm or to obstruction from a swollen mucosa and plugs of mucus, the patients recover on a careful diet and rational therapy. Both mild and severe cases occur, the latter often producing extensive gastrectasis. True hypertrophic stenoses are much more serious, and in the grave cases the result depends upon surgical treatment being undertaken early. To wait

too long is a grave error, and yet experience shows in many instances that proper variation of the food often produces excellent results and rapid cures. Koplik's cases bear out the importance of suitable diet. When dilatation and marasmus exist, the prognosis is more serious (Cautley). The milder cases have less hypertrophy and obstruction, and may get well with lavage and diet, or may pass into a mild chronic state with gastritis and ultimately die from marasmus. Mild genuine cases do exist, as in Cautley's patient, in whom the stenosis was apparently latent. There was severe wasting, and peristalsis was seen once. Such cases may terminate later in life with dilatation and hypertrophic stenosis, and perhaps originate some of the instances seen in adults. That congenital stenosis of the pylorus in adult life is common, as held by some recent surgeons, can scarcely be said to be proven.

The cases described by Maylard and others are not altogether convincing. Some had had previous hæmatemesis; several others showed no dilatation whatsoever, while the crucial test made by insertion of the finger through the gastric wound into the pylorus is not always a satisfactory method of testing for a true stenosis in that region.

Maier's series, reported in 1885, described autopsies on 21 patients—in all of whom the condition was regarded as congenital in origin despite the advanced age of many, up to eighty years, and even although there were often no secondary effects of a stenosis that had lasted many years. It may reasonably be asked if some at least of these were not rather of the nature of a pylorospasm as described by Pfaundler.

W. and J. R. Harper report a case that is remarkable chiefly on account of its recovery without operation. The chief symptoms were persistent vomiting of all food, starting two or three weeks after birth, with obstinate constipation, progressive marasmus, visible peristalsis and dilatation of the stomach, and a palpable tumor in the pyloric region. The successful treatment of this case consisted in the administration of small quantities of peptonized milk at frequent intervals, with a daily injection of half a pint of normal saline solution.

Treatment.—In the early stages the diagnosis is usually doubtful, and the patient should be subjected to medical treatment under careful observation, to determine whether one is dealing with stenosis from mere spasm, temporary obstruction, or with a true hypertrophic condition.

Dietetic.—Careful feeding with non-irritating food and rest after feeding is the main thing. If the child is being nursed, the breast feedings should be regulated. If artificially fed, a wet-nurse may be employed, or the simplest kind of food, as whey, albumin water, peptonized milk, raw meat juice, in small amounts, may be used. Fats should be very limited in quantity, these being the last to leave the stomach, and for this reason a milk with a low fat percentage should be selected. Heubner recommends feeding every three hours, as much as they care to take, letting them vomit, and applies poultices three times daily for two hours, changing every half hour. Batten suggested nasal feeding, but this is of doubtful value. The infant should be weighed from time to time to estimate the value of the feeding.

Mechanical.—Lavage is of great benefit, relieving the vomiting and distress, keeping up the appetite and helping nutrition indirectly. It may be done twice daily at first, before feeding, with a soft catheter, and should there be evidence of increased HCl, one may add sodium bicarbonate (1 to 250).

Later, once a week is sufficient. One must remember that lavage may seem to cure because it stops the vomiting, and thus deceive the clinician as to the true nature of the malady. Saline injections per rectum may be useful.

Medicinal.—Various remedies are suggested, the main objects being sedative: $\frac{1}{15}$ of a drop of tet. opii with a few drops of tet. valerianæ (Heubner); or $\frac{1}{100}$ gr. of cocaine or $\frac{1}{4}$ m. tet. opii, every hour (Cantley).

Alkalies by neutralizing the excess of HCl help to relax the spasm. They should be given after feeding.

General Medical Directions.—One must watch the weight, but need not expect a gain at once. If after a few days there is marked loss of flesh, weakness, tetany, or convulsions, operation is indicated. If there be persistent vomiting, progressive emaciation, visible peristalsis, and absence of faeces in the stools, the indications for operative treatment are obvious.

Operative Measures.—Just so soon as it seems wise to operate this should be done without delay. The prognosis in medical cases shows a mortality of 72 per cent. in 104 cases (but the diagnosis in these was not definitely enough classified). Surgical treatment shows a mortality of 56 per cent. in 39 cases.

Statistics, however, are unreliable, as much depends on the severity of the case, the underlying cause, and the technique of the operation.

Loreta's operation of divulsion is rather out of favor with some surgeons, on account of its danger and unreliability. It is not much less serious than gastro-enterostomy, and there may be need of repetition of the operation, or liability to starvation ensues; if in the divulsion the muscles are not ruptured, they will recontract. Sometimes the serous coat may be ruptured and a fatal peritonitis results. The statistics are, however, favorable; Burghard¹ in 16 cases had only 5 deaths. In 29 cases in the literature thus treated, 15, or over 50 per cent., recovered. Stiles, who succeeded in 4 out of 9 cases, prefers gastro-enterostomy as being more reliable, although even with this latter method his results have as yet not been very reassuring.

Gastro-enterostomy is a more lengthy operation and may have serious after-effects, yet is doubtless the operation of selection with most modern surgeons. One authority had 6 recoveries in 15 operations. In a total of 59 cases collected there are 29 recoveries, 49 per cent. (Thompson,² 1906). Stiles had 5 recoveries out of 10 cases, and 2 of these even died later from acute enteritis.

Pyloroplasty is difficult to perform. Symptoms persist for a few days after operation because the obstruction is not at once relieved, but this may be obviated by cutting out a wedge of muscle at the time of operation. After operation one should follow by rectal feeding every four hours for two days, every six hours for two days, and give sips of hot water for two days. Overfeeding may lead to enteritis. Dent³ had 7 recoveries in 11 cases; 2 died some months later.

Pylorectomy is too radical to be taken into consideration under ordinary conditions, and hitherto the mortality under this treatment has been higher than by any other of the operative procedures.

¹ *Transactions of the Clinical Society*, 1907, p. 122.

² *Journal of Surgery, Gynecology, and Obstetrics*, 1906, iii, p. 521.

³ Quoted by Stiles, *British Medical Journal*, 1906, ii, p. 947.

CIRRHOSIS OF THE STOMACH.

Definition.—This may be defined as a non-malignant, slowly developing, and progressive affection of the stomach, characterized by diffuse or circumscribed increase in the connective tissue, involving chiefly the submucosa, and to a less extent the other layers, and sometimes extending to the connective tissue in the parts bordering upon the stomach.

Nomenclature.—Although the condition has been recognized for more than two centuries, there have been so many views as to its underlying cause and its real nature that the synonyms for the disease are very numerous; *e. g.*, chronic interstitial gastritis; sclerosis of the stomach; fibroid induration; fibroid disease of the pylorus (Habershon); hypertrophy with induration (Cruveilhier); submucous hypertrophy; submucous sclerosis with chronic gastritis, and callous retroperitonitis (Hanot and Gombault); hypertrophic stenosing gastritis (Boas); hypertrophic stenosis of the pylorus (Lebert); fusocellular sarcoma (Lyonnet); plastic linitis (Brinton). This last term seems to have been that generally adopted, and certainly the French writers have done much to perpetuate its use. The nomenclature is probably also confused through an apparently unnecessary mingling of various conditions, each of which has a different cause. The names mostly describe the nature, situation, and extent of the lesion, the anatomical change being essentially the same in these cases.

The two following morbid anatomical entities appear to exist: (1) Simple sclerotic affections of the stomach, non-malignant in nature, affecting the submucosa chiefly, and usually the other coats of the organ to a greater or less degree. (2) Cancerous neoplasms with extensive secondary sclerosis of the stomach. These do not properly come into the category of primary plastic linitis, the fibrosis being merely secondary to the cancer. There may, or may not be, metastases. Sometimes the malignant lesion is obvious to the naked eye, while at other times it is only found after much laborious microscopic examination, the extensive interstitial new-growth so overwhelming the epithelial proliferation that it easily escapes detection. Hence arises the view that probably all instances of plastic linitis may be reckoned among the scirrhus cancers if one but takes the pains to find the true lesion. It would seem that in many articles dealing with the subject of gastric sclerosis these two entirely different conditions have been confused. To state that in an apparent case of plastic linitis cancerous structures have been found is merely to explain that the condition present is, after all, not one of simple plastic linitis, but in reality a true cancer associated with the sclerosis. While, undoubtedly, many cases of supposed sclerosis of the stomach are carcinomatous, yet sufficient careful observations have been made, both clinically and anatomically, to render it certain that a pure non-malignant sclerosis of the gastric submucosa does exist, as originally described many years ago by Andral and others.

Historical.—Apparently observations on this condition are at least several centuries old, and, according to W. H. Welch, date back to the seventeenth and eighteenth centuries, perhaps even earlier. At all events, Butzen, Lieutaud,¹ Pohl, Voigtel,² and other early observers have noted

¹ *Historia Anat. Med.*, 1779.

² *Handb. d. path. Anat.*, 1804, ii.

many instances of sclerosis of the stomach, although it is not until after 1830 that the observations become frequent. We are indebted to Andral¹ (1835) for the first accurate description of the condition and its differentiation from cancer. He noted the frequency of the pyloric situation, and observed that the orifice might vary in different cases—be normal, contracted, or dilated. In the same year Cruveilhier² defined the condition as “hypertrophy with induration,” and noted fungating excrescences of the mucosa, great thickening of the adjacent tissue, hypertrophy of the muscle, and its invasion by strands of fibrous tissue from the mucosa. The absence of juice on expression of the cut surface differentiated it, in his mind, from true scirrhus.

Monneret, Salse, and especially Broca³ (1850) drew further attention to the condition, the last-named author dilating on the peculiarities of its clinical history, so unlike that of ordinary carcinoma. It was about the same time that Gluge⁴ drew the attention of the Germans to it in their own country, describing a case with simultaneous involvement of the pylorus and cardia. One year later Brandt⁵ described his series in which a case occurred with an analogous growth near the cæcum, and Lebert reported one in which both the colon and the rectum showed processes similar to that in the stomach. To none of these authorities, however, did the existence of cancer seem probable until Rokitsansky, in 1859, maintained that submucous hypertrophies of the stomach belong in reality to the group of fibroid cancers. Brinton, however, two or three years later, gave to the condition its most used name “cirrhosis of the stomach,” and “plastic linitis,” thus assuring to it a clinical and anatomical distinctiveness. He classified it, not in the category of gastritis, because he held that the mucous membrane was never affected at the onset, and, indeed, was often quite intact; neither was it an hypertrophy, for often in the midst of new tissue much atrophy exists, but rather he regarded it as a cirrhotic inflammation, and not a pure sclerosis, which latter term applies merely to the last stages of the disease. Brinton recognized both its resemblances to and its dissimilarity from cancer, and admitted the difficulty of satisfactory classification owing to the insufficiency of data.

Hughes Bennett, Handfield Jones, Hare, Quain, and Wilks maintained, however, the original opinion of Andral as to the benign nature of the disease. Following upon these observations in the next two decades various writers endeavored to associate a number of causes with the disease. Thus, for example, to Niemeyer and to Leube, as well as Jaccoud, chronic gastritis appeared to be the main etiological factor. A primary atrophy appeared to be the cause according to Fenwick,⁶ while to Bouveret⁷ the pathological process resembled that seen in scleroderma. Huchard,⁸ in 1894, suggested arterial sclerosis as a cause, and Pedrazzini, in 1898, described it as a senile change. Still more recently Poncet and Leriche,⁹ in 1905, attached some importance to the presence of tuberculous infection. Among the most important papers which have appeared in connection with the subject was

¹ *Précis d'Anat. Pathol.*, 1829-1835.

² *Anat. Path.*, 1835.

³ *Bull. Soc. Anat.*, 1850, p. 207.

⁴ *Atlas d. path. Anat.*, 1850, ix.

⁵ *Die Stenose des Pylorus*, Inaug. Diss., Erlangen, 1851.

⁶ *On Atrophy of the Stomach*, etc., London, 1880.

⁷ *Traité des maladies de l'estomac*, 1893.

⁸ *Bulletin Médicale*, 1894.

⁹ *Bull. de l'Acad. de Méd.*, May 30, 1905.

that of Hanot and Gombault,¹ in 1882, a contribution of special value in view of the careful microscopic examination made in almost serial sections of the diseased tissue. The lesions were most marked at the pylorus and lesser curvature, and there was much invasion of the neighboring tissues, thus creating a callous retroperitonitis; but nowhere could they find either macroscopic or microscopic evidences of a malignant origin. Such, too, was the experience of Tourlet,² who carefully discussed the whole subject some ten years later, and who concluded from his own observations and from analyses of others that the disease was not cancerous. Nevertheless, there has been a tendency of late years to advance still more strongly the theory of its cancerous origin, and Garrett, in 1892, Bret and Paviot, in 1894, as well as Hoche, in 1903, strongly maintained this position. The cases described by them showed definite evidence of cancer, or of ulcers which had become malignant, and more or less malignant extension and metastases were observed. To Bret and Paviot cirrhosis of the stomach is merely a cancer with cells which are metatypical in the stomach, and the real nature of which is only seen in those rare instances where one finds in the generalized process the typical gastric epithelial cells which came originally from the stomach lesion. Garret, a pupil of Bard, seemed to regard the condition as of the nature of a fusicellular sarcoma.

In a recent communication by Schacher³ either a clinical or anatomical entity to this condition is denied. Stress is laid upon the microscopic similarity to cancer and the ever-increasing frequency with which, histologically, signs are found of the epithelial nature of the neoplasm; Brelet⁴ seems rather to coincide with that view (1905). Still more recently, however, Jannesco and Grossman⁵ insist on the benign character of the lesions (1908).

Pathogenesis.—Diverse as the views are of the nature of this condition, and uncertain as is still its origin, many of the theories advocated may be readily refuted by an analysis of the reported cases. That it is not essentially an advanced stage of chronic gastritis, for instance, is demonstrated by the comparative infrequency of typical inflammatory lesions of the mucosa, which in so many cases appears quite unaffected. The same holds true of the theory of Fenwick, for primary atrophy is by no means a constant or even frequent lesion. Arteriosclerosis and senile changes do not again explain many cases occurring in young subjects, devoid of the manifestations of senile disease. Nor, again, is tuberculosis anything but a rare accompaniment and obviously a mere coincident. The occasional presence of ulcer, as described by Formad,⁶ Hoche,⁷ and others, does not certainly explain the origin of those advanced cases of linitis in which, in spite of the extensive disease of the submucosa, the overlying mucous membrane was intact, and showed not the slightest evidence of either recent or healed ulceration. It is, however, more difficult to decide whether the theory of its cancerous origin is or is not applicable to all cases. There is no doubt that clinically the features are, as a rule, not usually identified with cancer of the stomach, *e. g.*, the long duration, the slow cachexia, the long remissions, the character of the emesis, etc.; furthermore, the presence of the oblong tumor,

¹ *Archiv. gén. de physiologie*, 1882, ix, p. 410.

² *Thèse de Paris*, 1902.

⁴ *Gaz. des Hôp.*, November, 1905.

⁶ *Journal of the American Medical Association*, 1887, vol. ix, p. 599.

⁷ *Rev. de Méd.*, 1903, vol. xxiii, pp. 944 and 1079.

³ *Thèse de Paris*, 1905.

⁵ *Revue de Chirurgie*, 1908.

cylindrical, regular, unyielding, with an absence of secondary invasion or metastases, all point to a condition of chronic inflammation rather than that of carcinoma. These cases described, in which a definite cancer accompanied the linitis, naturally are not to be considered in this obscure pathogenesis, being, as they are, instances of either primary or secondary carcinoma, and not necessarily implying that all the gastric cirrheses are of this origin, any more than does the occasional coincidence of cirrhosis of the liver with cancer prove that all such hepatic diseases are primarily malignant in nature. Hoche, who cites a case of ulceration with subsequent cirrhosis and final cancerous degeneration with metastases, merely describes a phase in pathological development which applies to any chronically diseased tissue, but does not prove that all cases of linitis develop in this way.

Conclusions from the observations hitherto made would lead one to believe that plastic linitis is an interstitial overgrowth, chiefly in the submucosa, with or without muscular hypertrophy, in some cases primary from an unknown cause, in others secondary, perhaps to ulcer or to cancer, or to a cancerous degeneration of an ulcer; and at other times perhaps associated etiologically with other irritating conditions such as might anywhere in the body favor the overgrowth of connective tissue.

Etiology.—Apart from what has already been said of the relation of other diseased conditions of the stomach to plastic linitis, very little may be added as to its etiology. It is essentially a disease of adult life, the average age being about forty-five years. Tourlet analyzed 33 cases according to the age: 3 occurred from twenty to thirty years of age, 9 from thirty to forty years, 7 from forty to fifty years, 7 from fifty to sixty years, and 7 from sixty to seventy years.

Sex seems to have no influence in its causation, although apparently a few more men than women are affected. Trauma, through compression of the epigastrium from occupation, is mentioned by Hare, Schacher, and also by Snellen as a possible factor. There is no proof that alcohol induces the linitis, nor does the frequent association of this poison with chronic gastritis bring it into any nearer relation to the other malady. Moreover, in many of the cases described there is an assured absence of an alcoholic history. Syphilis has been mentioned as a possible cause, although where this is present one may be said to be dealing with a specific disease by itself and not with plastic linitis.

Morbid Anatomy.—The lesions which are essentially sclerosis and hypertrophy vary in their location, in the extent and multiplicity of their various foci, and in the degree of invasion of other structures. The typical lesion usually described is that found in the diffuse variety. The stomach itself is either of normal size or contracted. It may be freely movable, fixed in the epigastrium, or pulled up underneath the liver by firm adhesions to the gastrohepatic omentum. While at times it is universally surrounded by adhesions, these are usually most firm at the splenic end. They may be so firm and extensive as to cause intimate attachment to any of the neighboring organs and tissues, to which the same process may likewise extend. The capacity of the stomach is usually diminished in these diffuse cases, and it may not contain more than 150 cc. Dilatation occurs, as a rule, only when the pylorus is involved. Externally the organ has a grayish appearance, with a curious opacity due to the overlying peritoneum. It is oval, irregularly ovoid, or cylindrical in shape, resembling in size and form a thick-

ened transverse colon. Its consistency is firm, in parts almost cartilaginous, but with the elasticity and resilience of a large artery. When removed from the body and placed upon the table its shape is retained because of the thick firm walls. On section it is firm, and the knife yields a grating sensation, while the slit gapes open like an artery. The cut surface is unevenly thick, and may be in some places twenty times greater than normal; although in most instances the average thickness has been 2 to 2.5 cm. Usually the greatest thickness occurs at the pylorus, the hypertrophy gradually diminishing as one gets farther away from the outlet of the stomach. On the other hand, in a few cases the cardiac end was the main seat of involvement (Formad, Lyonnet), and Petibon has described a case of cancer at the cardia with a secondary linitis most marked at that region. In other patients the lesser curvature is chiefly involved, or it may happen that several portions are separately diseased, the intervening tissues remaining comparatively normal. Everywhere the cut surface has a fibrous pearly grayish appearance, sometimes glistening like a tendon, or again resembling the chronic indurated cedema of subcutaneous tissue as seen on transverse section. The individual layers of the stomach are readily recognized even though the colors be somewhat similar.

The mucosa is often normal in appearance and structure, although at times it is slightly thickened and firmer than normal, and in some cases could be lifted off from the submucosa unless fibrous strands extending from below attached it intimately to the submucosa. The thickness is diffuse or patchy, and in a few instances areas of atrophy are also seen. As a rule, pale and anæmic, the mucous membrane nevertheless presents, here and there, patches of ecchymoses or pigmentation. It may be smooth or rough, the surface being sometimes thickly granular with excrescences, due, presumably, to the contraction of the interstitial tissue beneath. Sometimes a few polypi are seen. Attention has been drawn by various authors to the presence of ulcerations and erosions, especially at the pylorus (Hoche), the cause of which is ascribed by Pilliet to vascular obliteration due to fibrous constriction.

The submucosa presents the greatest hypertrophy and thickness. Its tissue is dense and firm, its color grayish white, and the fibrous strands running through and from it in all directions traverse the underlying muscle tissue so as to form a network about the individual bundles (hence the name "linitis").

The muscularis is likewise hypertrophied, although it is invariably less thickening than the submucosa. There is often a true hypertrophy of the muscle, as well as an increase in its apparent thickness due to the extra growth of connective tissue among its bundles. The fibrous tissue may finally invade even the subserous cellular tissue, so frequently, indeed, that Brinton regarded this layer as the site of origin. The peritoneum may or may not be involved; while sometimes normal, it would seem, in a few instances, to be more extensively involved than all the other tissues. Out of 47 cases, 9 presented a normal peritoneum, and 21 showed a chronic peritonitis. In the remaining 17 the peritoneal condition is not mentioned. In the case of Hanot and Gombault so great was the involvement of peritoneum as to render it worthy of a special title—*rétropéritonite calleuse*. There was chronic inflammation, with thickening, induration, retraction of the stomach, and fixation of the organ. Amid the adhesions which involve the gastrohepatic omentum, the

great omentum, and the gastrosplenic ligament, obliteration of the common duct and the portal vein has occurred, and even an obstructive biliary cirrhosis. The visceral capsules may be thickened (liver and spleen) and these organs themselves sclerosed or atrophied. Even the intestines are sometimes thickened (more especially the transverse colon), and their caliber diminished even to complete obstruction. Simultaneous involvement of the cæcum has been mentioned.

Amid the thickened tissues of the mesentery the lymphatics are frequently seen to be dilated, sometimes so markedly as to suggest that the lymphatic obliteration was the primary cause of the condition (Bouveret, Tourlet).

The glands are almost invariably small, although their cut surface may show an interstitial increase similar to that seen in the submucosa. Only in these cases, comparatively few in number, in which carcinoma was present, have the glands been enlarged, and such instances, of course, come under the category of epithelial neoplasms and not of simple plastic linitis.

Microscopic Examination.—The mucosa, as is to be expected, shows either a normal condition or the minute lesions of a chronic productive or atrophic gastritis. The glandular structures may be displaced and the development of fibrous tissue may cause constrictions and cystic dilatations of the tubules, or else may have caused the gland structures to entirely disappear. In a case mentioned by Osler the mucosa was smooth without any trace of glandular elements—a distinct sclerosis of the mucous membrane itself with atrophy. The lymphoid nodes are sometimes proliferated. The submucosa, which is much thickened by strands of fibrous tissue densely arranged, and forming a network, may be at times most abundantly gathered around the bloodvessels. And the muscularis, which is normal in some places, is in others like an irregular checker-board traversed by intersecting lines of fibrous tissue. The subserous layer exists in various stages of progressive inflammation, and the peritoneum itself may show increased connective tissue, and more or less destruction of the endothelial cells.

The circumscribed form, of which the commonest and most typical lesion, is the “hypertrophic stenosis of the pylorus,” shows anatomical lesions of a similar nature, although, as a rule, there is dilatation of the organ rather than contraction, and the muscle tissue itself gradually degenerates. Habershon’s fibroid disease of the pylorus and Boas’ hypertrophic stenosing gastritis are the synonyms. The delimitation toward the stomach itself is usually gradual, while toward the duodenum it is much more sharply defined. The cases of congenital hypertrophic stenosis of the pylorus may sometimes belong to this category, although their origin is more of the nature of a developmental morbidity than of an inflammation. Of the other sites of localization the cardia has been already mentioned, and Formad’s case, in which the greatest thickening was at that orifice, is worthy of special mention. An ulcer existed in the posterior wall near the cardia, the floor of which was formed by splenic tissue and the peritoneal coat of the transverse colon. The stomach was contracted and held less than 150 cc.

Symptoms.—The onset is insidious, and the early development slow, gradual, and by no means easily determined. The early symptoms correspond in most particulars to those of chronic gastritis, and there are vague symptoms of indigestion, with a fickle appetite and usually anorexia, a sense of heaviness after meals, and later vomiting of mucus or of partially

digested foodstuffs. In the early stage pain is moderate or entirely absent, and rarely so severe as to suggest ulceration. For one or more years these milder symptoms, which are usually slowly progressive, or else with occasional long periods of remission, are gradually superseded by signs that indicate a more serious disease. The pains, which at first were insignificant, become more constant, of a dragging nature, sometimes spontaneous, sometimes only excited by food. They may extend from the epigastrium to the back, and are usually increased by pressure. Nausea, eructations of gas, and vomiting of liquid gastric contents accompany the pain, which is usually relieved by free emesis. The vomiting, which is always digestive in time, appears sometimes at once after meals, as though from an involvement of the oesophagus, and Roux has drawn special attention to the diagnostic importance of this symptom as suggesting gastric intolerance because of the rigid contracted cavity. When the chief lesion is at the cardia, regurgitation of food or mucus would be a common sign, but in the commoner diffuse cases the vomiting appears one to two hours after meals. It is most continuous in the severe cases, and if stenosis of the pylorus be present there will be signs of retention. In about 20 per cent. of the cases slight hæmatemesis is noted, but there is never much bleeding because there is rarely ulceration. For the same reason obvious melæna is uncommon. The increasing limited capacity of the stomach for food is an important symptom. The intestines are not commonly involved, but sometimes the bowels are irregular and there may be a mucous enteritis. The general appearance of the patient may vary; while, as a rule, some pallor is conspicuous, the patient has lost flesh, and has become somewhat cachectic, yet at other times, both the nutrition and the color are well preserved, a circumstance to which Trousseau has drawn special attention.

Examination of the abdomen in uncomplicated cases may show some retraction of the epigastrium, or a mass, more or less localized, may be seen prominently beneath the abdominal wall or moving with respiration. Peristalsis may be visible. Palpation demonstrates, usually, a diffuse resistance in the epigastrium, not always marked, especially if the contracted stomach be drawn up and fixed beneath the liver. At other times a hard oblong transverse tumor may be felt in the epigastrium, and extending to the left false ribs, or again only in the neighborhood of the pylorus. The mobility of the tumor varies according to its site and the nature and extent of any adhesions present. It has been mistaken for a movable spleen and for the transverse colon. Trousseau has drawn attention to the sensation of grating conveyed to the palpating hand on deep respiration. As time progresses, the condition may extend beyond the stomach to the peritoneum and the surrounding viscera. Ascites may develop, make palpation difficult, and conceal the real underlying cause; or the symptoms may change to those of intestinal obstruction, in which to the conditions of cachexia and anasarca may be added persistent and uncontrollable vomiting until death follows from asthenia. Jaundice is rare in these cases, and apparently due only to constriction of the common bile duct from adhesions. When the cardiac orifice is mainly involved, so that constriction occurs, the process is more rapid and the fatal termination induced through starvation and inanition. Rapid emaciation, dysphagia, regurgitation of mucus and food are the characteristic features. When typical hypertrophic stenosis of the pylorus is present the signs are those of a benign stenosis. To the primary digestive

symptoms are added the slow development of the affection, the marked emesis, which is digestive, infrequent, and small in amount at first, and later less often but more abundant; later come the signs of marked motor insufficiency with constipation, great thirst, and hunger, diminution in the quantity of urine, localized pain, visible peristalsis, perhaps a visible tumor, rapid emaciation, and death from inanition.

Analysis of the Gastric Contents.—The chemical findings are as yet too few and variable to be regarded as pathognomonic. Gabbi¹ records the examination of vomitus after a Leube test meal (meat, bread, and water). Hydrochloric acid was absent and there were traces of peptone. Lactic acid was present in abundance. Rosenheim, in an examination of the gastric contents obtained identical results. Chaput and Pilliet,² however, found hydrochloric acid present in one instance. Boas, in his description of hypertrophic stenosing gastritis, asserts that the hydrochloric acid is diminished or absent, that lactic acid is abundant, as are also Boas-Oppler bacilli. Sarcinæ were absent in all the cases examined. Tourlet describes hypersecretion in one of his cases. There was no free hydrochloric acid, but traces of lactic acid were present.

Diagnosis.—This has rarely been made during life. When, even at autopsy, it is so difficult to make an anatomical diagnosis, one can scarcely expect that the clinical diagnosis would be easy. While the uncomplicated cases are difficult to determine, still more do the cases with complications lack ready means of diagnosis. The chief features are the long duration, the slow cachexia, the limited capacity of the stomach, and its intolerance of much food at any one time, the absence of hæmatemesis or mælena, the presence of an oblong cylindrical tumor in the epigastrium in patients in whom the history and physical signs present neither the local evidence of carcinoma, metastases, or any signs of generalization. Plastic linitis has frequently been mistaken for cancer of the stomach; likewise, on account of the ascites and the history of alcoholism, for portal cirrhosis; and on one occasion for obstructive biliary cirrhosis. Simple chronic or tuberculous peritonitis resembles it on account of the peritoneal pain, the evidences of ascites, and the signs of intestinal obstruction. When pyloric stenosis is present one considers the possible presence of an old gastric ulcer and consequent perigastritis, and for the elucidation one trusts to the past history. In deciding on the possible presence of cancerous degenerative changes in a gastric ulcer one considers the two periods in the history—that of the ulcer and that of the malignant transformation. Inflammatory bands from the peritoneum, and, more rarely, the presence of adenomata, or local connective-tissue tumors, or spasm of the pylorus, must be considered. In a record by Bouillaud a toxic gastritis caused by nitric acid was followed by extensive hypertrophy. Syphilis rarely presents similar symptoms; as a rule, the lesion presents mere strands of fibrous tissue, and gummata are uncommon. Nevertheless, much interest attaches to Einhorn's case, as reported by Brissaud, in which a large tumor, the size of a pigeon's egg, disappeared in a few weeks under antiluetic treatment. Deguy,³ in 1896, described his case in which, for the first time, a diagnosis was made during life, and subsequently confirmed at autopsy. McCrae⁴ reported a similar experience from Osler's clinic.

¹ *Riforma Med.*, 1893, vol. iii.

² *Bulletin de la Soc. Anat.*, 1896, p. 154.

³ *Bull. Soc. Anat.*, Paris, 1896.

⁴ *Johns Hopkins Hospital Bulletin*, 1901, p. 25.

Course and Duration.—Chronicity is the characteristic feature, and although Bouveret's case lasted apparently only twenty months from the onset, this is much shorter than the average duration. Tourlet's patient suffered seven to eight years before operation, and the average is perhaps ten to fifteen years. No general rule can be made, for the duration depends on the variety of the lesion. Circumscribed cases obstructing neither orifice will naturally last much longer than those which are localized at the pylorus, or still shorter time if at the cardia. The diffuse cases will be of still shorter duration than those confined to the pylorus, and where peritonitis, ascites, and adhesions supervene, the course is rapidly and progressively downward. In Schacher's case it was thought that a pelvic operation precipitated a rapid decline and an extension of a cancer co-existent in the inflammatory tissue.

Treatment.—The rational treatment is essentially surgical. Medically there are but two considerations: in the early stage to treat the condition as in chronic gastritis, and later, where stenosis exists, to employ palliative treatment until the more radical cure is attempted by surgical measures. Gastro-enterostomy as a mere palliative operation has been found successful, but where possible, a removal of the diseased tissue, in view of the possible cancerous nature of the affection, is the one thoroughly scientific treatment worthy of consideration—for the localized condition, pylorectomy, and for the diffuse lesion, total resection.

Mauclaire and also Terrier have recorded cases in which an apparent cure followed exploratory laparotomy, and Roux¹ records an interesting case in which the patient was well three and one-half years after operation (anterior gastro-enterostomy). Total resection was performed by Gayet and Patel² (1904) for a linitis of definite malignant origin, this being probably the first operation of its kind for this condition.

HÆMATEMESIS.

Synonyms.—Gastrohemorrhagia; vomitus cruentus.

Definition.—Hæmatemesis is a vomiting of blood—regardless of its origin—and it must not be confounded with gastrorrhagia, for the blood vomited in hæmatemesis may come from other sources than the stomach itself. It is merely a symptom, to be referred not only to the stomach itself, but also possibly to conditions in structures contiguous to that organ. It is much more common than is usually recognized, probably because spectroscopic, microscopic, or chemical aids are not usually employed to reveal the presence of blood when not in macroscopic quantity. Certain minute hemorrhages more often go unrecognized than is usually believed, and in this way the early manifestations of disease remain undetected.

There may be no anatomical lesions (diapedesis), as in Lancereaux's³ supposed neuropathic hemorrhage occurring in gouty, nervous, or rheumatic individuals; or the lesion may be so minute as to escape detection even although the hemorrhage be very copious. Such conditions often arise from a general oozing from various portions of the mucous membrane, and in

¹ *Rev. méd. de la Suisse Rom.*, 1905.

² *Arch. gén. de méd.*, March, 1904,

³ *Journ. d. Med. Interne*, 1906, x, p. 73.

some cases the blood streams forth from a wide surface of the mucosa. More often the lesion is obvious (rhæxis), and some gross anatomical disturbance, such as ulcer, cancer, or phlegmon, explains its origin.

Etiology.—The bleeding may be (1) gastric, or (2) extragastric.

1. Gastric Bleeding.—Gastric bleeding may be from *local* or *general causes*.

Local Causes.—These may be *direct* or *indirect*.

Direct Local Causes.—Among these are: Ulcers—simple, round, tuberculous, typhoidal, diphtheritic, septic, etc.; varices and miliary aneurisms, perhaps associated with arteriosclerosis; gastritis, simple, acute, chronic, phlegmonous, or toxic; cancer and trauma. The trauma may be direct or indirect, and the causes physical and chemical.

Physical causes, *e. g.*, foreign bodies; the strain of vomiting; injury from the stomach tube; stretching from distention by CO_2 . Chemical causes, as poisons, purgatives, emetics with degeneration of the mucous membrane, fatty, hyaline, or amyloid; postoperative hæmatemesis, as yet ill understood.

Indirect Local Causes.—Portal obstruction in cirrhosis of the liver, syphilis of the liver, atrophy, pressure on the portal vein, portal thrombosis (of these cases, 80 per cent. are œsophageal in origin). Thoracic disease with circulatory obstruction, emphysema, chronic pleurisy, fibroid lung, and organic heart disease. Splenic enlargement, as in splenic anæmia, etc.

General Causes.—In these hemorrhage occurs from various mucous membranes as well as from the gastric. The conditions are many: sepsis (with or without signs of local lesion); the exanthemata (hemorrhagic forms) and other fevers such as typhoid; hæmatemesis neonatorum perhaps belongs to this group; autotoxic states, uræmia, cholæmia, etc.; blood dyscrasias and diseases, hæmophilia, purpura, scurvy, pernicious anæmia, leukæmia, etc.; possibly degeneration of vessels and capillaries, or emboli with hemorrhagic infarctions or infected thrombi; neuropathies, epilepsy, tabes, general paresis, hysteria, and meningitis, in all of which the hemorrhages may be vasomotor in origin; vicarious menstruation and burns.

Debove and Courtois-Suffit attribute these unexplained hemorrhages without discernible lesions to abdominal congestion due to depressor nerves acting on arterial tension, as has been declared possible by Ludwig and Cyon.

2. Extragastric Bleeding.—Apart from the hemorrhages which arise in the œsophagus from varices, etc., the blood may come from abscesses contiguous to the stomach bursting into that organ; or ulcers and fistulæ may open up a suppurating tract into the stomach and cause hæmatemesis. Vertebral caries or aneurism of the abdominal aorta or cœliac axis may perforate the stomach and cause hæmatemesis.

Age and Sex.—The age at which hæmatemesis occurs is usually from fifteen to forty; it is rare in infants. It is probably more common in women.

Morbid Anatomy.—General and local signs may be described. The general signs depend on the degree of hemorrhage. In fatal hemorrhages there is general pallor of the viscera from depletion, and if the bleeding be slow or recurrent there may be fatty degeneration of the heart muscle, liver, kidneys, gland cells, and the alimentary mucosa. The originating cause too, will in large part determine the nature of the general changes, *e. g.*, cachexia in cancer, cloudy swellings in sepsis, etc. The mucous membrane of the gastro-intestinal tract is usually pale, and above the seat of the

hemorrhage there will be the remains of blood more or less tarry. The clot formed varies in appearance. If there be little blood and gradual oozing one gets a chocolate or coffee-ground appearance perhaps, with black specks like powder sprinkled through the contents, or a diffuse bloody, or darkly stained fluid. At the seat of the hemorrhage one may find eroded vessels, and sometimes an imperfect clot formation. The opening is often very difficult or impossible to find or only evident after injection from the main vascular arrangement.

Symptoms.—These depend chiefly on the amount of blood poured out and the rapidity with which it flows. Sometimes the gastric hemorrhage is latent, especially when the hemorrhage is small, and the blood must then be detected in the vomitus or fæces as occult hemorrhage. At other times the blood is not vomited even when very copious, and the patient may even die with no other signs than those of internal hemorrhage. In true hæmatemesis the blood, however, is vomited and may be large (up to two liters) or small in quantity. When small in amount it is usually mixed with mucus and food and appears either as minute specks or as streaks mixed with the other gastric contents. Its color depends upon its amount and the time in the stomach. The blood of minute hemorrhages is most easily transformed in color. When fresh, the color is bright and the blood cells are unchanged. When for some time in the stomach, the blood is dark and the oxyhæmoglobin is changed to hæmatin, resembling coffee-grounds. Experiment has shown that if 50 cc. of blood reach the stomach and remain in it for five minutes, the red cells become altered and the color already much darker than normal. Microscopically one sees cells more or less broken down and blood pigment. Sometimes, again, there is much dark-red clot with an acid reaction.

Subjective Symptoms.—The patient may have a sense of warmth in the epigastrium and along the œsophagus; sometimes an abnormal pulsation with fulness is felt in the abdomen. Pain is frequently felt in the epigastrium for some time before hemorrhage, and may cease when this occurs. If quickly vomited, the blood may come not only through the mouth, but from the nose, and may get into the air passages and cause coughing. As a rule, hæmatemesis is accompanied by great anxiety and fear of death. The general signs of hemorrhage are present—pallor, faintness, dizziness, ringing in the ears, specks before the eyes, dimness of vision, cramps in the thighs and calves and perhaps clonic spasms, œdematous swellings of the smaller bones and eyelids, drowsiness, insomnia at night; the pulse is often dicrotic, anæmic systolic murmurs are heard over the heart, and a venous hum over the large vessels. The blood is pale, the red cells and hæmoglobin are reduced. The individual cells are pale, showing poikilocytosis, and many are nucleated. Leukocytosis frequently occurs. The elementary bodies are present in clusters, probably associated with the blood plates and broken-down white cells. Blood changes are more especially found in recurring hæmatemesis. Fever is present, especially if the intestines have contained much blood, because absorption occurs; on this account the temperature is not infrequently reduced by purgative treatment.

Albuminuria is rare, but may occur. The mouth is dry; there is fœtor of the breath and great thirst; the skin becomes scaly, and the hair may fall out. Delirium, dim vision, amaurosis of unexplained cause, sometimes optic neuritis and neuroretinitis, with white spots on the retina, may be all present.

Recovery is sometimes rapid, at other times very slow, because bleedings from the vessels may be often repeated and small in quantity.

Diagnosis.—When hæmatemesis has occurred one must consider the following questions: (1) Does the vomitus really contain blood? (2) If so, is it from the digestive tract? (3) Is it gastric in origin, and if so, does it come from near the cardiac end, from the duodenum, or from the stomach proper? (4) What is the etiological and anatomical lesion? (5) What vessel is affected? (6) Is it amenable to medical treatment, or, if not, is it accessible to operation?

Hæmatemesis is so often latent or unrecognized that one must make careful observations both as regards the previous gastric history and the present state of the abdomen and gastric contents or vomitus. Not infrequently there is a fulminating gastrorrhagia, in which no blood is vomited and the diagnosis is made from the general sensations and symptoms of hemorrhage and the previous history of gastric disease. Even when very copious it may not be evacuated and the patient may die with merely the signs of internal hemorrhage, or vomiting may be delayed for some time, or the blood may only appear in the stools.

1. *Is Blood Really Present in the Vomitus?*—It not infrequently happens, both with patients and physicians, that a faulty diagnosis is made because the food previously taken may resemble blood, *e. g.*, red wine, cherries, cranberries, red cabbage, red turnips, red sausages, coffee, and cinnamon. Or pseudohæmatemesis may be associated with various preparations of iron or of bismuth; the bismuth crystals, moreover, under the microscope, resemble to some extent those of hæmatin. In doubtful cases one should always examine microscopically for blood cells or blood pigment, and where doubt still exists, one should test with the spectroscope or by chemical means, to prove the presence of these so-called occult hemorrhages.¹

2. *Is the Blood from the Digestive Tract?*—The blood may not be from the digestive tract at all, but from hæmoptysis, epistaxis, or from a ruptured aneurism. To differentiate from hæmoptysis one must remember that in hæmatemesis the blood is dark red in color, usually resembling venous blood; that it contains no air, has usually an acid reaction from contact with the gastric juice; that it is mixed with food; that it is vomited, not coughed; that it does not contain tubercle bacilli, for tuberculous ulcers of the stomach are extremely rare; and lastly, local findings in the lungs are absent, while those in the stomach are often obvious; or there may be signs of a heart lesion, the possible cause of hemorrhagic infarction of the lungs. Epistaxis may give rise to error through swallowing of the blood and subsequent vomiting. For this reason it is always well to examine the nasal cavity carefully and to look for polypi and other source of nasal hemorrhage. The same may occur in empyema and cardiac disease.

Aneurisms which leak gradually may simulate true gastric disease, as

¹ The most recent test for occult blood, and an accurate one, is that devised by Meyer, of Munich. Two grams of phenolphthalein, 20 grams of potassium hydrate, and 10 grams of zinc dust are boiled in 100 cc. of distilled water until the mixture is completely decolorized. It is then filtered and used as the reagent. Equal parts of the reagent and of hydrogen peroxide are then mixed in a test tube, and to this mixture the fecal solution to be tested is added after having been boiled and cooled, as in the benzine test (p. 194). A positive reaction is indicated by the production of a carmine to pink color. It is said to be sensitive 1 to 1,000,000.

in a remarkable case recorded by Janowski,¹ whose patient entered the hospital after vomiting one liter of blood and fainting. Several weeks previously he had complained of a burning dry sensation in the œsophagus, and for three years had had pain in the left hypochondrium. He had been in hospital nine days when the tube was passed after the administration of a test meal, and one minute later he died. An aneurism of the aorta had perforated the œsophagus and been ruptured by the passage of the tube.

3. Having excluded other sources, one must decide *whether the hemorrhage be gastric, duodenal, or œsophageal.*

Esophageal Varices.—The œsophageal veins empty into the azygos veins, which, in their turn, are tributaries of the vena cava superior and inferior respectively. When portal obstruction occurs there is sometimes overflowing of the vena cava inferior and therefore of the hemi-azygos and azygos veins, and through these of the œsophageal veins. There is nothing distinctive about the hemorrhage from the œsophagus. Esophageal varices are most commonly associated with early cirrhosis of the liver, and one should therefore examine carefully for it. The evolution of the condition may help the diagnosis. In two-thirds of such œsophageal hemorrhages the patient improves, but recurrence takes place after intervals of days, months, or years. Sometimes they are fulminating in character. The etiology of cirrhosis, *i. e.*, alcoholism, etc., will aid the diagnosis, as also will the physical examination of the abdomen. *Esophageal cancers* may also simulate gastric ulcer, but bring with them other evidences of malignant disease. *Esophageal ulcer* causes pain after swallowing, increased secretion and regurgitation of mucus and food, and progressive signs of stricture.

Duodenal hemorrhage from ulcer is sometimes accompanied by hæmatemesis. Men are more commonly afflicted. As a rule, the stools show the first evidence of this condition. The pain is usually situated more to the right; it occurs later after meals than does that of ordinary gastric ulcer (two to four hours), or, at all events, is increased at that time; the digestive signs of pain, etc., coming on several hours after meals and the presence of HCl help to establish the diagnosis.

4. *The nature of the causative lesion* is often most difficult to determine, and is sometimes impossible. *Neuropathic hæmatemesis* is a problematical condition which is perhaps receiving less recognition each year; it is presumably more common in nervous people, in females than in males, comes on after emotions with prodromata of heat, weight, or pain in the epigastrium and sometimes vertigo. It co-exists or alternates with hemorrhages from other organs which are presumed to have a similar cause. It is not usually fatal, and seems to have no effect upon the system in general. As a rule, patients thus afflicted pay but little heed to the hemorrhages when they occur. One should look for the other stigmata of hysteria. In all such cases, however, the exclusion of ulcer is not easy, and one will do well to diagnose the more serious anatomical lesion until further proof of a mere neuropathic origin is possible.

Vicarious hemorrhages usually occur in the absence of any gastric signs, have more or less periodicity, and at best are quite rare. They occur either at the time of the menses, or at times quite apart from that period. The menses may be absent and replaced by the hæmatemesis, or the latter may

¹ *Ztschr. f. klin. Med.*, 1902, xlv, p. 43.

coincide with diminished menstrual flow. Legroux had a patient in whom monthly hæmatemesis occurred during the first seven months of pregnancy.

Varices in the stomach are very rare, and doubtless have the same causes as those in the œsophagus. Letulle found only two cases in sixteen years.

Venous Stasis.—Although varices are rare, dilatation of veins is common, and a rupture of the capillaries of the mucous membrane frequently occurs, as in cirrhosis of the liver, inflammations or tumors of the pancreas, cancer of the gall-bladder, wounds in the vicinity, etc. In the vomitus of peritonitis and intestinal obstruction there may be a little showing of blood; perhaps, however, these types are infectious in nature.

Hemorrhagic Erosions.—These show only bleeding and few or no other symptoms. Often they are only found in the washings from the empty stomach and usually then associated with chronic gastritis. The use of the term to indicate a special disease as suggested by Einhorn is disputed by Elsner and others. The bleeding may occur by the mouth or the rectum, as in latent ulcers; they are rarely fatal, and usually occur in men who are in poor condition, with advanced tuberculosis, or alcoholics with cirrhosis, etc. C. H. Miller¹ attributes many gastric hemorrhages to erosions from swollen or ruptured lymphoid follicles in the stomach mucosa which has become thinned, while the basement membrane is unusually thickened. Thus the follicles are less covered, and under abnormal conditions, many lymphoid follicles become swollen, disintegrated, and softened. They in this way reach the surface and may burst through the mucosa, thus leaving the basement membrane temporarily exposed to the gastric juice, which digests some of the numerous vessels of the submucosa. Hemorrhage then occurs.

Miliary aneurisms of the submucous arteries as a part of generalized aneurisms cannot be distinguished clinically.

Simple Ulcer of the Stomach.—Hæmatemesis coming on in healthy individuals or in those who have suffered from stomach disorders is due in the majority of cases to ulcer in some stage. The history with the symptom complex, the oft-recurring hemorrhage in youthful subjects with good appetites and good albumin digestion, the presence of HCl in the contents, and the absence of cachexia aid in determining the presence of ulcer. The important point is not so much the exact diagnosis of the lesion, as the diagnosis of the value and possible success of intervention. The hemorrhage is often single and fulminating, as happens with the type of cases known as "exulceratio simplex." The term should become obsolete in the light of modern experience.

Cancer of the stomach is usually easy to diagnose from the history and the findings. The patient is older, with anæmia, perhaps cachexia, anorexia, no HCl in the contents, but lactic acid instead, etc. While sometimes hemorrhage in cancer is acute and fatal, as a rule, bleeding is small, recurrent, and of a coffee-ground appearance. Occult blood in the stools is much more constant. However, many exceptions occur, and all possible factors must be considered together.

5. *What Vessel is the Source?*—This cannot usually be told from the amount of blood present, the nature of the attack, or the evolution of the disease. Experience teaches that the most copious bleedings may sometimes arise without any evidence of anatomical lesion, and, vice versa, small

¹ *British Medical Journal*, 1906, p. 1547.

continued hemorrhages may occur even when the aorta is the direct source of the blood. Cases are on record where a cardiogastric fistula occurred, with hemorrhages lasting for a number of days prior to death. Similar instances are published of fistula between the aorta and intestines. Perhaps the large arterial trunk most commonly involved is the splenic artery, and yet here, also, where ulceration into it had occurred, sudden death followed in only 3 out of 17 cases. When the bleeding occurs suddenly from an artery of moderate size, the hemorrhage may be copious and emesis immediate; in such a case the blood is usually bright in color, at all events much more so than in slow oozings from small capillaries where one obtains merely coffee-ground admixture of blood, mucus, food, etc. So few minutes are, however, required to alter the color of the blood when in the stomach, that the bright blood is much less frequently found than in hæmoptysis.

Considering the possibility of copious hemorrhages from invisible sources, the frequency of minute bleedings from large trunks, and the changes occurring in the blood when in the stomach, one can appreciate the difficulties of determining whether the source be capillary, venous, or arterial.

6. *Are the Ulcer and Vessel Accessible for Operation?*—Fresh ulcers usually erode the superficial vessels in the wall and are readily reached; old ulcers may be adherent to the pancreas and the liver and are sometimes hollowed out. Adhesions form beyond the limits of the organ and are deeply situated, fixed in immobile tissues, perhaps hard or friable the vessels are deeper, less accessible, and often larger. In a word, they erode rather the extrinsic arteries, such as the splenic, and make operation difficult, perhaps impossible.

Prognosis.—This depends chiefly upon the cause and, to a less degree, upon the severity of the hemorrhage. In serious general conditions hæmatemesis is one of the worst prognostic symptoms of the disease. When blood flows from outside structures into the stomach the prognosis is bad. In poisoning cases the amount of hemorrhage itself is of no prognostic importance unless the contents be putrid, in which case a deeper ulceration may be suspected and possibly death from sepsis or perforation. It is often difficult to differentiate between rupture of the small and the large vessels, and statistics vary very much because there is nothing in the evolution of the condition or in the antecedent hemorrhage which can prognosticate a recurrence or progressive gravity or origin; one cannot say from the amount of blood and the area of pain what vessel is affected or whether the hemorrhage will recur. The most copious hemorrhages may have no visible anatomical lesions of the mucosa. Large hemorrhages, however, are rarely fatal (3 to 4 per cent., Lebert).

Sometimes in gastric ulcer hemorrhage causes cessation of all the other signs; in cirrhosis of the liver that organ often diminishes in size after a hæmatemesis. The cause is often more serious than the hemorrhage itself. In ulceration the prognosis is difficult to determine. Hyperacidity after the hemorrhage may act as an irritant and cause renewed ulceration and bleeding.

For one month after a severe hemorrhage the condition continues serious, but from that time onward becomes less so, so far as life is concerned, but there is always the same uncertainty as regards recurrence. After eight to ten days the prognosis may be given as good, because of thrombus formation, although one must always keep in mind the possibility of hemorrhage from another part of the ulcer where a newly attached vessel has simultaneously opened up. In cancer, hæmatemesis may usually be considered as a fatal

sign because indicating advanced disease, although exceptions occur with bleeding at an early stage.

Preble¹ concludes, from an analysis of 60 fatal cases of hæmatemesis with subsequent autopsies, that: (1) While not rare, fatal hemorrhage is at all events not common in cirrhosis of the liver. (2) The first hemorrhage in cirrhosis is fatal in only one-third of all cases. (3) Œsophageal varices are the source of the bleeding in 80 per cent. of cases; in half of these, ruptures were found and are probably much more common than is generally realized. (4) Fatal hemorrhages sometimes occur from invisible ruptures of numerous capillaries of the gastric mucous membrane, and, as a rule, there are some accompanying signs of early cirrhosis of the liver.

Treatment.—The indications are as follows: (1) Prophylaxis, when the possibilities of hæmatemesis are recognized. (2) Discovery of the etiological factor. (3) Control of the hemorrhage as quickly as possible. (4) Prevention of its recurrence. (5) Prevention of injurious sequelæ.

Prophylaxis depends upon the discovery of the possible underlying condition and its judicious treatment. Control of the hemorrhage requires rest in general and of the stomach in particular. The patient should be put to bed with the head low and kept as quiet as possible. All tight clothing should be removed and the patient encouraged with the explanation that hæmatemesis of itself is of no very serious import. Should faintness supervene, the face should be sprinkled with cold water, ammonia placed beneath the nostrils and if necessary camphor oil, ether, or caffeine, given hypodermically; a hot bottle may be placed to the feet. In the most serious cases an intravenous saline may be given and the arms and legs bandaged. An ice-bag may be suspended from a cradle so that it will come into gentle contact with the epigastrium. All food should be stopped for three days and the patient should be allowed only some ice to suck, but not to swallow. After three days, nutrient enemata may be given, three times daily. No food should be given by the mouth until after the first week, and then, only gradually, boiled milk, broth, or gruel.

Lavage of the stomach is best avoided, so, too, all local hæmostatics. General hæmostatics are perhaps beneficial; adrenalin, in doses of 25 drops of the 1 to 1000 solution by mouth daily has apparently been of service. One may use rectal injections of calcium chloride, 4 to 8 gm. (5j to 5ij) daily, or gelatin is sometimes useful when given by the mouth. The reflex effect of heat as administered by hot rectal injections (115°) has been much recommended.

Accessory Treatment.—Cold water enemata are useful for retained blood in the bowel. For the anemia, which may persist, one should give good food, and iron if the patient bears it with ease, but its use requires great care.

Surgical Interference.—*Indications.*—The blood count and estimation of hæmoglobin per se are of little value. Appreciation of the general condition is more important than an estimate of the hæmatological state. If a low blood count has supervened on a previous chlorosis the condition is more grave. The pulse per se has doubtful value. The amount of hemorrhage is also of little importance as compared with recurrence of hemorrhage, which implies an end to medical treatment. For general surgical indications see the section on the Treatment of Gastric Ulcer.

¹ *American Journal of the Medical Sciences*, 1900, cxix.

CHAPTER VI.

DISEASES OF THE INTESTINES.

By ALFRED STENGEL, M.D.

PHYSIOLOGY OF THE INTESTINAL TRACT.

UNFORTUNATELY our knowledge of the physiology of the intestines has not yet proven of much practical value in the study of intestinal diseases or their treatment. The newer physiology of digestion (both gastric and intestinal) gives promise, however, of some practical applicability in the near future, and for this reason requires to be kept prominently in mind.

Motor Functions of the Intestines.—The movements of the intestines serve three important purposes: (1) The onward movement of the contents; (2) thorough mixture of the chyme with the digestive secretions and close contact of the contents with the absorptive mucous membrane; (3) the propulsion of the venous blood and chyle away from the bowel. The old idea that the main purpose of peristalsis is to cause the movements of the intestinal contents toward the rectum has been disproven by recent investigations, which show that certain conspicuous movements of the bowel serve the purpose of thoroughly churning the contents and thus mixing the chyme and the digestive fluids and at the same time bringing the contents of the bowel into more complete contact with the mucous membrane.

Three forms of movement are recognized: (1) Peristalsis proper; (2) rhythmic segmentation; (3) antiperistalsis.

Peristalsis Proper.—Peristalsis proper, or the movement that causes the forward flow of the bowel contents, is a wave-like contraction that runs for some distance, gradually diminishing toward the end of its course, where a new wave starts up. The bowel contracts behind the contents and relaxes in front of them (Starling). These movements vary in rapidity and strength under normal conditions, and may become excessive in pathological states. Violent stimulation may cause a tonic contraction of a segment or a considerable portion of the bowel. The peristalsis of the large intestine causes an alternate protrusion and retraction of the haustra.

Rhythmic Segmentation.—In skiagraphic studies of the movements of the intestinal contents Cannon found that constrictions occur at various places, and the portions of the bowel between the constrictions may subdivide by secondary segmentation. The segments subsequently re-unite and divide with renewed contractions. As many as thirty segmentations a minute were observed. These segmentations involve the contents of the bowel and do not indicate the kind of contractions that occur in the wall between the segments. Possibly the swaying movements or waving movements described by Raiser may be the occasion of the segmentation. These are backward and forward contractions and relaxations affecting a limited part of the bowel and running in a direction parallel to its length. There is no appreciable narrowing of

the lumen of the bowel. Such contractions might very readily divide the contents of the bowel into segments. Rhythmic segmentation continues during sleep, but may be inhibited by excitement. These movements cause a churning of the contents of the bowel and perhaps to some extent aid peristalsis proper in their onward movement.

Antiperistalsis.—Cannon has found this to be the important motor phenomenon of the large intestine. When the contents of the ileum are emptied into the large intestine they are carried forward for some distance. Antiperistaltic waves then begin and run upward to the ileo-caecal valve. A new significance is thus made manifest for the valve. The antiperistaltic waves working toward the closed valve churn the fluid contents and promote absorption by the mucosa. From time to time proper peristaltic contractions move the contents farther toward the rectum. Some investigators like Nothnagel have not been able to demonstrate the antiperistaltic waves, but the more modern methods of Cannon doubtless give more trustworthy results.

Mechanism of the Movements.—The movements of the bowel are caused by the irritating effect of their contents: solid, liquid, or gaseous. When empty, the intestines always are quiet. The irritation of the contents may be mechanical or chemical. Indifferent substances may act by their temperature or mere bulk. Of gases, hydrogen, oxygen, and nitrogen are indifferent, while carbon dioxide, sulphuretted hydrogen, and the hydrocarbons stimulate peristalsis.

Englemann believes the stimulus is transmitted through the bowel from muscle cell to muscle cell, but Nothnagel insists that nervous influences are always necessary, as do Gad and Lüderitz. These nervous stimuli originate in the plexuses of Auerbach and Meissner. Bayliss and Starling believe the swaying movements are myogenic in origin.

External Nervous Mechanism.—Stimuli are carried to the intestine through the vagus, and irritation of this nerve causes movements of the whole small intestine and upper half of the large. Irritation of the splanchnic nerves inhibits movements.

Cannon, however, has recently found that section of the splanchnics or of the vagi and splanchnics disturbed normal peristalsis very little. Other recent observers, like Bach and Ehrmann, found that one splanchnic nerve is the motor nerve of the longitudinal fibers and the inhibitory nerve of the circular fibers, while the vagus stimulates the circular and inhibits the longitudinal. Bayliss and Starling deny any motor functions to the splanchnic.

The lower half of the colon and rectum are innervated through the inferior mesenteric plexus and hypogastric plexus.

Pathological Peristalsis.—In pathological conditions various modifications of peristalsis may be met with. These may consist simply of excessive contractions, but in some cases rolling movements (Nothnagel) cause a contracted loop of the bowel to rotate like a wheel and then relax, while in other cases tonic contractions of a segment of bowel cause a stiffening and narrowing of this portion, which may last some time before it slowly relaxes. Complete cessation of all movements may result from emotion, fright, fear, etc., and is a common condition in certain diseases, such as peritonitis or infarction from mesenteric embolism.

Varieties of Pathological Peristaltic Movements.—(1) Increased peristalsis; (2) tonic contractions; (3) antiperistalsis.

Increased peristalsis may occur: (1) When the bowel contents are abnormal; (2) after the administration of purgatives; (3) after thermic or chemical irritation; (4) after emotional shock; (5) in neurasthenia; (6) from inflammations; (7) in cases of stricture of the bowel. Pathologically increased peristalsis may not differ in character from the normal, but in some cases "rolling" movements (Nothnagel) are observed. In these cases the loop of contracting bowel may perform active rotation somewhat resembling the rotation of a wheel.

Tonic Contractions.—When the intestine is empty these convert the tube into a solid cord of pale color. When a large part of the bowel is affected the abdomen may be scaphoid, as in certain cases of lead colic.

Tonic contractions of a loop filled with contents may cause violent pain. Ordinary and even excessive peristalsis alone does not produce pain.

Pathological antiperistaltic movements are found only in the large intestine according to Nothnagel. Others, however, believe that such waves may affect the small intestine as well.

Greutzer showed that fluid containing free particles introduced into the lower bowel may find its way high up, even to the stomach in a few hours. This upward movement seemed to occur most actively when the fluid was a sodium chloride solution. It did not occur with potassium chloride or hydrochloric acid solutions. Nothnagel had previously shown that sodium chloride applied to the external wall of the bowel causes upward movements running over several centimeters. Hemmeter has shown that an upward movement of particles may go on while the central mass of the intestinal contents is moving downward, and he believes the epithelium and muscularis mucosa are operative in this process.

Visible Peristalsis.—Normal peristalsis is visible only in exceptionally thin subjects. The movements of the large intestine can never be seen in health. In various diseases, notably in intestinal obstruction, active peristalsis may be seen through the abdominal walls.

Chemical Processes Occurring in the Intestine.—The digestive processes that take place in the intestinal tract are effected by secretions from the glands and lining cells of the intestinal mucous membrane itself; but, to a still greater extent, by secretions poured into the intestine from the liver and the pancreas. The important secretions are: (1) The pancreatic juice; (2) the bile; (3) the intestinal secretion or succus entericus.

The Pancreatic Juice.—This alkaline liquid is a thin and somewhat watery fluid, having a specific gravity of about 1007.5. The amount varies greatly, and cannot be stated with any certainty. In one case in which a permanent fistula existed (Glaessner), the amount was from 500 to 800 cc. per diem; but there is reason to believe that the fluid may not have been a pure pancreatic secretion. The secretion of pancreatic juice begins soon after food has been taken into the stomach, and reaches its maximum during the second to the fourth hour after that time. The character of the food influences, to some extent, the amount of secretion and the time of its maximum flow; and the rate of secretion seems to be especially dependent upon the promptness with which ingested food is discharged from the stomach into the duodenum.

The mechanism of pancreatic secretion has recently been determined by physiologists. It was first shown by Dolinsky, in 1895, that acids brought into contact with the mucous membrane of the duodenum promptly excite

pancreatic secretion. This effect of the acid is certainly to a large extent independent of any influence upon nervous mechanisms, since the result occurs after section of the vagus and splanchnic nerves, quite as readily as when these nerves are intact. Bayliss and Starling have demonstrated the probable mode of action. They found in the mucous membrane of the duodenum an enzyme, *prosecretin*, which, under the influence of a 0.4 per cent. solution of hydrochloric acid, is converted into *secretin*. The latter is absorbed and, reaching the pancreas through the blood, stimulates it to activity. It is not improbable that the HCl of the gastric contents may also exercise some effect upon the secretory and nervous mechanisms of the intestinal mucous membrane, but this is not of considerable importance. That secretin is carried to the pancreas in the blood has been proven by the fact that the pancreas may be stimulated by hypodermic injection of secretin.

The ferments of the pancreatic juice are: (1) The proteolytic ferment, trypsin; (2) a diastasic ferment, amyllopsin; (3) a lipolytic ferment, lipase or steapsin.

Trypsin.—It has been claimed by some that this is not a single body; but that a variety of trypsin, of different stability, occur. It has also been shown recently that trypsin is secreted in the form of a pro-enzyme, trypsinogen, which is inoperative as a proteolytic ferment until activated or converted into trypsin by an enzyme derived from the duodenal mucous membrane known as enterokinase. It is claimed by Bayliss and Starling that the trypsinogen cannot be converted into trypsin except through the action of this duodenal ferment.

Trypsin acts upon proteids in alkaline, slightly acid, or neutral media. Albumins are dissolved by its action without swelling, and, except serum albumin, are converted first into a globulin insoluble in water; later, into peptones soluble in water. Trypsin probably, however, carries the hydrolysis of certain peptones still farther, producing leucin, tyrosin, and other simple bodies. According to some recent observations, the prolonged action of trypsin destroys all peptones, leaving only end-products of a simpler nature.

Amylopsin.—This enzyme is similar to, if not identical with, the ptyalin of the saliva. It hydrolyzes starches, with the formation of maltose and achroödextrin. These substances are further acted upon by the maltase of the intestinal secretion, and converted by this into dextrose. According to Hoffmeister, the maximum amylolytic effect occurs in the presence of a moderate acidity.

Lipase or Steapsin.—This ferment was discovered by Bernard in 1849. It has never been isolated. Lipase splits neutral fats, with the formation of fatty acids and glycerin. The fatty acids thus liberated unite with alkalis to form soaps, the latter being further utilized for the emulsification of other fats. The recent experiments of Kastle and Loewenhart indicate that, by reversed action, lipase causes a synthesis of the products of the splitting of fat, with the re-formation of the original fat. This synthetic action is believed to take place in the intestinal wall, as well as in the tissues. Lipase is a widely distributed enzyme, being found in the blood and in many tissues.

Bile.—The quantity of bile secreted by the liver varies considerably under different conditions. The maximum secretion occurs about an hour after a meal. Proteid foods increase the amount of secretion; fats reduce it; carbohydrates appear to have little effect. Starling believes that the secretion

of bile is initiated by the action of a hormone (analogous with secretin) formed by the action of the acid gastric contents on the mucous membrane of the intestines.

The total quantity of bile made in twenty-four hours varies from 500 to 600 cc. (Ranke, Wittich). Hammarsten found similar figures in his observations of seven cases of biliary fistula in man.

The bile is a clear, alkaline, mucoid liquid, of yellowish to brownish color. Its important constituents are bile-acid salts (glycocholates and taurocholates) and pigments (bilirubin, biliverdin, etc.). The mucoid character is due to the presence of a nucleo-albumin or true mucin. Various other less important substances are present. There are also ferments, diastasic and lipolytic, which supplement the action of the pancreatic secretion.

The principal action of the bile in digestion and food absorption is its relation to the emulsification of fats. The alkalies contained in the bile unite with the fatty acids to form soaps, and the bile directly aids in the splitting of the fats.

It has been held that the bile exercises some antiseptic effect on the intestinal tract; and it probably has some influence in stimulating intestinal peristalsis.

Succus Entericus.—The secretion of the intestinal mucous membrane is the product of various tubular glands of the small intestine. The quantity of the secretion has been estimated by Prigl as being as much as three liters a day. It is probable, however, that the amount varies greatly in different cases and under different conditions.

Recent investigations have shown in the intestinal secretion and in the mucosa of the small intestines a number of important enzymes, of which the following have been most thoroughly worked out: enterokinase, erepsin, inverting enzymes, and secretin.

Enterokinase.—Enterokinase has been found in the mucosa of the duodenum, and seems to be the ferment that activates trypsinogen, converting it into trypsin.

Secretin.—Secretin is a stable body existing as a prosecretin in the intestinal mucous membrane. Under the influence of the acids of the gastric contents the latter is converted into secretin, which is absorbed, and reaches the pancreas through the circulation, stimulating this organ to activity.

Erepsin.—Erepsin completes the hydrolysis of proteids, acting upon the deuterio-albumoses and peptones prepared by the preliminary stages of digestion.

Inverting enzymes convert disaccharids into monosaccharids.

Besides these substances, a secretin has been thought to exist which originates in the intestinal mucous membrane and, after absorption, stimulates the liver to activity.

ABSORPTION OF FOODS IN THE INTESTINE.

The products of digestion are mainly absorbed by the bloodvessels, and, entering the portal system, are carried to the liver. Fats, however, after their cleavage in the intestines, are resynthesized in the intestinal mucous membrane and absorbed through the lacteals.

Carbohydrates are absorbed as simple sugars, monosaccharids, the disac-

charids being converted into the simpler form by the action of inverting enzymes in the small intestine. When an excess of sugar is administered there may be direct absorption without such preliminary change.

Fats are in part split into their constituents, fatty acids and glycerin, and in part emulsified, after which absorption through the mucous membrane occurs. The cleavage products are synthesized and, with the emulsified fat, enter the lacteals.

Proteids are absorbed after digestion by pepsin, trypsin, and crepsin; and are absorbed as peptones or proteoses. To some extent, however, simpler bodies, such as leucin, tyrosin, arginin, etc., may be formed, and may be later synthesized to reconstruct the albumin of the tissues. It has been claimed that proteids may be absorbed directly, that is, without preliminary digestive alteration.

Absorption in the Large Intestine.—To some extent, products of digestion that have escaped absorption in the small bowel are absorbed in the large intestine. Under normal conditions the main absorption that occurs in this part of the intestinal tract is that of water; and, as a consequence, the fluid contents of the upper part of the large intestine become more and more concentrated and finally solidified as they approach the rectum.

THE INTESTINES AS EXCRETORY ORGANS.

The importance of the intestines as excretory organs is not perhaps as yet fully recognized. It is well known that in certain diseases large quantities of serum may be poured into the bowel, carrying toxic matters of various sorts, chemical, bacterial, etc. Certain inflammatory, ulcerative, and necrotic processes in the mucosa doubtless result from such excretions. Investigations with certain drugs and chemical bodies of other sorts have given direct proof of this excretory function of the intestines. Among other substances, iron, calcium, strontium, lithium, and cæsium have been found to be largely excreted through this channel.

AUTOMATIC MECHANISM IN INTESTINAL PROCESSES.

The entrance of chyme into the bowel and the secretion of various digestive fluids is regulated in an automatic manner. When the gastric contents reach a certain degree of acidity from the presence of free hydrochloric acid a stimulus is exercised which occasions the opening of the pylorus and the expulsion of the acid chyme. This next acts upon the intestinal mucous membrane, and occasions a stimulus which effects the closing of the pylorus. At the same time, the acid acting upon the mucosa of the duodenum liberates secretin and possibly a hormone which stimulates the liver. To a minor degree, perhaps, the acid excites nervous stimuli, which aid in promoting pancreatic and hepatic activity. The alkaline bile and pancreatic juice entering the duodenum reduce the acidity of the chyme, neutralize it, and render it alkaline, thus establishing conditions favorable for the action of the various ferments. When the acidity of the duodenal contents has been neutralized, the stimulus which caused the closing of the pylorus is removed and a re-opening of the pylorus with discharge of more chyme follows.

The inert trypsinogen of the pancreatic juice is converted into trypsin by the ferment enterokinase, which like secretin is liberated from the intestinal mucosa under the influence of the acid chyme. The other ferments derived from the intestinal mucosa are quite possibly secreted as a result of the same cause. The further course of digestion and absorption in the small and large intestine is determined by the continued action of the digestive secretions and by the activity of the movements of the bowels. These, as has been shown, are largely the result of stimulation by the contents.

THE FÆCES.

The fæces consist of undigested and indigestible residues of food, together with altered digestive secretions, certain excretions from the intestinal mucosa, and a notable percentage of bacteria. According to the recent investigations of Strasburger and others, not less than a third of the dried substance of the feces consists of bacteria, and in some pathological conditions the amount is even greater than this.

The quantity of faecal matter passed depends upon a variety of conditions. The amount of food, its digestibility, the absorptive power of the intestines, the quantity of bile or pancreatic juice, or of mucus thrown off by the intestinal glands, and the activity of bacterial processes in the bowel all influence the quantity of faecal matter. Age, occupation, or habits of life, and functional conditions or diseases influencing the degree of peristalsis also exercise a decided influence. On a mixed diet, 120 to 150 grams, representing from 30 to 37 grams of solid residue, are passed in twenty-four hours. The greater the quantity of indigestible residue, as in the case of certain vegetables, the larger the faecal output. The variation of amount in different individuals is very considerable, as is shown in a table of Harley and Goodbody,¹ who found among 118 cases variations from 30 to 282 grams with a mixed diet. The average was 102.8 grams.

The color of the fæces is largely dependent on the character of the food. With a mixed diet the color is a light or dark brown, and is largely due to urobilin. Large amounts of meat cause a darker color of the stools, on account of the presence of altered blood pigment, hæmatin, and sulphide of iron. The greenish color seen when an abundance of green vegetables is eaten is due to chlorophyl. The fæces passed by persons whose diet consists largely of milk are light yellow, the light color being caused by the quantity of fat in the excreta. Bright-green stools in children or adults may be due to bacterial pigments or to the passage of unaltered bile. Sometimes there is a light color resulting from the presence of bubbles of gas mixed with the faecal matter. The light color of fæces in certain liver diseases is due to absence of bile and its derivatives. (See Acholic Stools.) Some medicines, especially iron, bismuth, lead, and silver, cause black stools; while others, like senna, rhubarb, and hæmatoxylon, cause a yellowish or reddish color.

The odor of the stools is due to skatol and volatile fatty acids. In fermentative and putrefactive conditions various aromatic bodies may impart a sour to putrid odor. Sometimes all trace of odor is wanting.

The consistency of the stools is normally dependent upon the amount

¹ *The Chemical Investigation of Gastric and Intestinal Diseases*, 1906.

of water, fat, and mucus associated with the insoluble residues. Lack of digestion and absorption of fat or excess of fat in the diet, excessive water drinking, abnormal excretion of mucus from the intestinal mucosa, and increased peristalsis which hurries the contents of the bowels through the large intestine so fast that the normal absorption of water and consequent inspissation cannot take place, all tend to make the stools soft, semiliquid, or even watery. In certain pathological conditions (choleraic diarrhoeas) there is an abundant watery outpouring into the bowel, and large watery evacuations are the result. The stools are more firm than normal when the food contains but little indigestible residue or when in the various forms of constipation the contents are retained too long a time in the large intestine.

The reaction of the normal stools is usually alkaline or neutral. Sometimes, however, a faint acid reaction is met with. Diet and pathological conditions of the bowel may occasion more decided acidity in some cases.

The chemical composition of the faeces varies widely. The amount of water in the faeces of persons on a milk diet was found to vary from 73.29 to 79.32 per cent., and in persons on a mixed diet was on the average 75.72 per cent. (Harley and Goodbody). Of the organic substances met with normally, fats and their derivatives, soaps, albumin, mucin, phenol, indol, skatol, fatty acids and their salts are important. The daily excretion of nitrogen in fasting persons has been found to vary from 0.17 gram to 0.446 gram per day, the average being 0.254 gram (Schmidt and Strasburger). With a mixed diet Harley and Goodbody found a discharge of from 0.30 to 1.55 grams per day, the average being 0.97 gram. Excess of diet, especially such as contains a large amount of residue, causes progressive increase in elimination of nitrogen. The percentage absorption of nitrogen of the food, with various forms of diet, varies from 90 to 96 per cent. Approximately, one-half of the nitrogen of the faeces is contained in the bodies of the bacteria. Fat, like nitrogenous substances, is excreted in the faeces even when the patient is fasting. The total excretion per day under these circumstances is from 0.57 to 1.21 grams (Schmidt and Strasburger). With ordinary diet, either milk or mixed, the daily excretion is from 3 to 7 grams, the proportion of absorption varying from 91 to 98 per cent. The character of the fats ingested, and especially the melting point, determines to some extent the degree of absorbability. Phenol, skatol, and indol in the faeces are products of bacterial processes, and their amount is to a large degree dependent on the activity of such processes. As derivatives of these substances are largely absorbed and excreted in the urine, the examination of the latter is utilized as a measure of the activity of such decompositions in the intestinal tract.

Among the inorganic constituents of the faeces are chlorides and carbonates of alkalies, phosphates of the alkaline earths, and other inorganic salts in small amounts. The amount and source of the calcium salts have excited special interest. It is generally believed that calcium is to an extent eliminated by the large intestine, although some investigators deny this.

Microscopic examination of the faeces serves to discover undigested particles of food, such as meat fibers, connective tissue, and starch bodies, that may be significant of different forms of intestinal or gastric disease. In inflammatory conditions mucus, pus cells, epithelium, and blood corpuscles may be found, and occasionally portions of exfoliated mucous membrane or portions of tumors of the bowel may be recognized.

Mucus may be found as gelatinous or shred-like particles or as considerable masses, visible to the naked eye, or as stringy formations visible under low powers of the microscope. Mucus from the upper bowel is often colored yellow by bile pigments; that from the colon or rectum is grayish or white. Certain small masses of gelatinous material that resemble mucus on superficial examination, presenting a yellowish color, may prove to be albuminous substances, and small globular masses, like frog spawn, are found to be derivatives of vegetable matter of the diet. Among the crystals found in normal fæces triple phosphates are the most common. Crystals of neutral phosphate of lime, and of oxalate of lime, cholesterin and Charcot-Leyden crystals, may also be found.

Acholic, Colorless, and Fatty Stools.—Acholic stools are yellowish gray or actually white in color. To a large extent this is due to the absence of urobilin, the normal pigment which is a derivative of bile pigment. In any condition in which complete obstruction of the common bile duct or of the hepatic duct occurs the fæces become of the color described. The biliary obstruction at the same time causes jaundice. Sometimes, however, the same kind of stools occurs without jaundice and without obstruction of the bile duct, as has been shown by Bamberger, von Jaksch, Walker, and others. In these cases the light color is usually due to the presence of enormous quantities of fat which may be dependent upon disease of the pancreas, obstruction of the pancreatic duct, or to various diseases of the intestines which interfere with the absorption of fat. Chemical analyses have shown in these cases that urobilin is present, although its color is obscured by the excess of fat. Another explanation of colorless stools in the absence of diminished outflow of bile is that the bile pigments are converted into colorless substances (leuko-urobilin). In these cases the stools give reactions for bile, and may be devoid of the excess of fat before referred to.

Colorless stools may occur independently of any disease of the liver or pancreas. In various forms of intense diarrhœa, as in cholera, dysentery, or certain cases of enteritis, in tuberculous peritonitis in children (Berggrün and Katz), and occasionally in various other diseases, the fæces may be quite colorless. A diminished excretion of bile may play some part in these conditions, but in most cases the altered color is the result of the amount of unabsorbed fat present in the stools. Sometimes, when a milk diet is continued for some time, the stools become almost white for the same reason, and nearly always this form of diet causes a lighter color than normal.

Fatty Stools.—From what has just been said it will be seen that various conditions may occasion excess of fat in the stools. As a rule, however, the term fatty stools is applied to cases in which masses of fat can be seen in the fæces or in which there is a distinctly oily appearance of the excreta. When somewhat watery, oil may collect on the surface of the liquid. Microscopically, fat crystals in large numbers are discovered.

Fatty stools may result from: 1. Increased ingestion of fat, as in persons consuming large quantities of milk, cream, olive oil, or cod-liver oil.

2. When absorption of fat is interfered with (*a*) by diseases of the bowel, such as enteritis, ulcerations, amyloid disease, etc., or (*b*) by active peristalsis and the prompt evacuation of the upper bowel.

3. When bile is excluded from the bowel by obstructions of the bile duct. The excess of fat associated with acholic stools has been mentioned.

4. When disease of the pancreas or obstruction of its ducts causes defi-

ciency of pancreatic juice. In these conditions the most typical fatty stools are met with. A veritable fatty diarrhoea may occur. The association of obstructive biliary conditions often aids in the production of the fatty stools of pancreatic disease. A difference between the fatty stools of biliary obstruction and of lack of pancreatic secretion has been indicated by Müller. In the former condition a large percentage of the fat present (84.3 per cent.), had suffered cleavage, whereas in cases of lack of pancreatic fluid only 39.8 per cent. of the fat had become converted. The passage of large amounts of undigested meat fibers, and the association of certain other clinical evidences may indicate the existence of pancreatic disease, but fatty stools in themselves do not suffice to establish such a diagnosis.

Intestinal Sand.—Occasionally gritty, sand-like material is discharged from the intestines. This usually consists of granular particles of grayish or reddish color, and on chemical examination has been found to contain various organic and inorganic substances. Duckworth and Garrod found, in a case in which chemical analyses were made, the following constituents: water, 12.40; organic matter, 26.29; inorganic matter, 61.31. The inorganic constituents consisted of calcium oxide, 54.98; phosphorus pentoxide, 42.35; carbon dioxide, 2.20; residue, containing traces of magnesium and iron, 0.47. Analyses by other investigators have given similar results (Harley and Goodbody). The term intestinal lithiasis has been applied to this condition, in the thought that there is a particular form of catarrh of the intestines with the discharge of calcareous matter comparable to the formation of calculi in other situations.

Intestinal sand is probably formed in the colon, as it usually contains considerable urobilin and little bile-pigment. It must, therefore, be produced in a part of the intestines in which the conversion of bile-pigment has progressed considerably. The condition with which intestinal lithiasis is most commonly associated is mucous colitis, but it is sometimes found independent of this condition.

A condition (false intestinal sand) that requires differentiation from the discharge of intestinal sand is that in which various residues of vegetable food are passed. These may have a sandy character that suggests true intestinal sand.

BACTERIOLOGY OF THE INTESTINAL TRACT.

Enormous numbers of bacteria normally occupy the intestinal tract of man and animals. A conception of the number may be gained from the knowledge that Strasburger has found in accurate investigations that one-third of the weight of the dry residue of the faeces consists of the bacterial bodies. In normal adults the weight of the dried bacteria passed in twenty-four hours is as much as 8 grams; while in certain dyspeptic conditions it may reach 14 to 20 grams. When habitual constipation is present the weight falls to 5.5 or as low as 2.6 grams. Even the last figure, however, represents enormous numbers of organisms. Strasburger has estimated the daily output at 128,000,000,000.

Distribution of Bacteria in the Intestines.—Billroth recognized that there are far more bacteria in the large than in the small bowel. Systematic examinations show a progressive increase in number from the duodenum to the large intestine. The duodenum and jejunum may be

entirely free six hours after meals (Cushing and Livingood). The use of sterile food and care of the mouth for a few days before operations on the duodenum and jejunum enabled these investigators to secure a sterile condition of these parts of the intestinal tract. Below the jejunum bacteria are probably never absent after birth.

The kinds of bacteria as well as the number vary in different parts of the digestive tract. This is doubtless due to changes in the conditions in different situations. The gastric secretion, the bile, and the pancreatic juice have probably only a moderate effect in destroying bacteria ingested with food. Smith and Tennant claimed that the secretions of the intestinal mucosa have an inhibitory effect on the multiplication of bacteria. The condition of the intestinal contents with reference to the process of digestion and the presence or absence of oxygen are other factors that have an influence on the number and kind of organisms present in different localities. Herter has shown that the conditions are practically anaërobic below the middle of the small intestine, which accounts for the presence of anaërobic forms in the lower bowel.

The organisms generally found in the duodenum and jejunum are various forms of micrococci and other varieties swallowed with food or saliva. In the lower part of the small intestine are found the obligate intestinal bacteria which belong respectively to the groups *Bacillus lactis aërogenes*, *B. coli*, and *B. bifidus communis*. These forms remain as permanent inhabitants and are not dependent on ingestion with food. Although capable of exercising pathogenic properties under certain circumstances, they are ordinarily so adapted to the organism and it to them, that their presence is devoid of any pathological effects. An evidence of their adaptation to the organism is seen in the fact that the serum of normal individuals has little agglutinative influence on these organisms. Although within the intestinal tract, they are practically outside the body, and whatever products are elaborated by their growth apparently have little effect on the system. The most abundant of the three forms of microorganisms named is the colon group. It is usually held that these are most abundant in the large bowel, but Cushing and Livingood found the maximum just above the ileocæcal valve. Their number decreased decidedly in the colon.

Among the forms met with in the lower small intestine and in the large bowel are certain anaërobic forms, notably *B. putrificus* and *B. aërogenes capsulatus*. Recently considerable attention has been given to the possible pathological results of the growth of such forms (Metchnikoff, Herter).

Many other microorganisms have been found in the intestinal tract under seemingly normal conditions. Most of these are closely allied to one or other of the forms alluded to. Matzuschita isolated 44 varieties of bacteria from 48 specimens of fæces.

Influence of Age on the Kind and Number of Bacteria.—The meconium before birth is sterile. Very soon after birth microorganisms are swallowed or enter the anus, and a considerable flora is soon present. In nurslings the *Bacillus bifidus* is the most abundant form in the large intestine, the *Bacillus coli* being less numerous. In bottle-fed children the dominant form, as in adults, is *B. coli* (Herter).

In adults and the aged the putrefactive organisms are more common, and, as a rule, putrefactive processes in the intestinal tract are more readily developed and more intense.

The Significance of Bacteria in the Intestinal Tract.—Are bacteria in the intestines to be regarded as a normal condition and necessary to life? Nuttall and Thierfelder kept young guinea-pigs, that had been removed from the maternal uterus by Cæsarean section under aseptic precautions, for some weeks in a sterile condition by feeding sterile food and allowing only sterile air to reach them. The animals gained weight and in every way developed as well as the controls. It has been claimed by Levin, but disputed by Chauveau, that polar bears in the Arctic region have no bacteria in their intestines. On the other hand, conclusions the reverse of those of Nuttall and Thierfelder have been arrived at by some other investigators, who found that animals did not develop in a normal manner when kept free of bacteria (Schottelius, Madame Metchnikoff, Moro). Pasteur and Nothnagel believed that bacteria are essential to physiological life, and certainly, whether essential or not, they are universally present and exercise a distinct effect on the intra-intestinal processes both physiological and pathological.

The role of bacteria in the conversion of bile pigments into urobilin, the final decomposition of products of proteid digestion by bacteria, the possible presence of enterokinase of bacterial origin (Dellezenne, Breton), and other evidences of the part played by bacteria in what appear to be purely physiological processes may be recalled.

Under ordinary conditions the bacteria of the fæces are practically all dead. Klein estimated that little over 1 per cent. are living forms, and Strasburger found even a lower proportion. The causes of this practical sterilization of the fæces have not yet been determined with certainty. Possibly the changing reactions and other conditions at the different levels of the bowel may play some part, as may also influences exerted by the bowel wall and its secretions. Perhaps products of the bacteria themselves occasion their own destruction.

Defensive Role of the Intestinal Bacteria.—There is considerable experimental and clinical evidence to prove that the normal intestinal flora is useful in protecting the individual against accidental pathogenic invaders. There seems to be little doubt that *B. coli* and *B. bifidus* exercise some inhibitory action on the growth of pathogenic organisms such as the putrefactive anaërobes and various specific pathogenic forms. Conradi and Kurpjuweit attribute this effect to antibacterial substances elaborated by the *B. coli*. They believe, also, that the same substances destroy the organism itself, thus accounting for the nearly sterile condition of the fæces. Lactic acid formed in the intestinal tract or swallowed with food may exercise a restraining influence on the growth of pathogenic (putrefactive) organisms. This may explain the controlling influence of a milk diet observed clinically in cases of intestinal putrefaction.

Conditions Influencing Increased Growth and Virulence of Intestinal Bacteria.—Various digestive disturbances may increase the growth of bacteria by causing larger amounts of nutritious material to pass down to the lower bowel. In habitual constipation, when practically all absorbable material is taken up in the small intestine, the number of bacteria in the fæces is the lowest met with under any conditions. The total bacterial output was found to be as low as 2.6 grams as against as much as 20 grams in dyspeptic conditions and 8 grams in normal persons (Strasburger). Excessive feeding acts in the same manner as digestive disturbances, and in

part as a result of the dyspeptic conditions caused by it. Catarrhal conditions of the bowel, the result of excessive multiplication of the bacteria or occurring independently, favor further increase of bacterial growth and increased virulence of the organisms. When ulcerations, erosions, or other lesions of the mucous membrane are present, the ordinarily harmless intestinal bacteria may gain access to the tissues and may occasion local or general infections. Sometimes a symbiotic relationship is established between different bacterial forms or between bacteria and protozoa like the *entamoeba coli*. The *Bacillus coli*, for example, is said to grow in virulence in the presence of staphylococci and streptococci (Coco).

Pathogenic Organisms in the Intestinal Tract.—Among the important organisms of certain pathological significance are the tubercle bacillus, the typhoid bacillus, and the various forms of the paratyphoid and paracolon groups, the dysentery bacillus, the cholera bacillus, streptococci and staphylococci, and the *Bacillus botulinus*. The *Bacillus coli* in some cases undoubtedly acts in a pathogenic manner within the intestinal tract, for some cases of systemic infection, as well as inflammatory conditions of the gastro-intestinal tract, are occasioned by this organism.

Other less frequent invaders sometimes cause pathological results. Among these may be named the *Bacillus pyocyaneus*, various micrococci, and probably certain anaërobic organisms like the *Bacillus aërogenes capsulatus*, the *Bacillus bifidus*, and the *Bacillus putrificus*. Certain rare forms of bacterial infection, such as glanders, anthrax, actinomycosis, and leprosy, may be mentioned here. Their discussion is appropriately considered in other volumes.

Clinical Results of Bacterial Invasion of the Intestines.—Leaving out of consideration the definite infections of the intestinal tract, certain types of intestinal and systemic derangement may be recognized as resulting from invasion of the intestinal tract by different forms of bacteria which do not occasion special diseases. Herter (*Bacterial Infections of the Digestive Tract*, 1907) has recently classified these in three groups: (1) The indolic type of intestinal putrefaction; (2) the saccharobutyric type of chronic intestinal putrefaction; (3) a combined type.

The indolic type appears to be occasioned mainly by the action of the colon bacillus group, although other organisms may play some part. The colon bacillus alone cannot break up native proteids, and must, therefore, act in conjunction with the digestive secretions or putrefactive bacteria, such as, *e. g.*, *B. putrificus*. This type of intestinal putrefaction is frequently associated with obstruction of the bile duct and pancreatic diseases, as well as with occlusion of the small intestines. Herter also calls attention to its occurrence in cases of marantic children. The most distinctive diagnostic feature is the increase of ethereal sulphates in the urine, the proportion of these to fixed sulphates being from 1 to 7 up to 1 to 4. Intestinal distention, weakness, irritability, and other nervous manifestations are met with in these cases.

The saccharobutyric type appears to be due to the action of anaërobic organisms, especially the *Bacillus aërogenes capsulatus*. The *Bacillus putrificus* may, however, also be associated with it. Gaseous distention and intestinal irritation following the use of carbohydrate foods, and the passage of light-colored stools with great excess of gas, are among the clinical indications. The condition occurs more commonly in adults than in children,

and occasions general weakness, various nervous symptoms, and sometimes high grades of anæmia approaching the type of pernicious anæmia. Loss of weight and general loss of vigor are not unusual.

The third type, representing a combination of the other two, presents also a combination of the clinical features. The distinction largely depends upon the examination of the fæces and the association of high grades of indicanuria. Nervous symptoms are apt to be early in appearance and conspicuous; and later, anæmia becomes a striking feature. The patient finally falls into a condition of chronic invalidism.

Treatment of Bacterial Invasions of the Intestinal Tract.—The problem of disinfecting the gastro-intestinal tract has occasioned much discussion, but still remains practically unsolved. The most important element in treatment is the proper regulation of diet and the avoidance of all infection of food. This involves the avoidance of all putrefactive contamination of food by proper care in its preservation and preparation, and the withdrawal of such articles as cheese, uncooked fruit, or milk. The careful cleansing of the mouth is also an important element.

The quantity of food consumed is of importance. Excessive eating occasions the passage through the intestinal tract of increased quantities of unabsorbed material and favors bacterial multiplication. A strict regulation and limitation of the diet is, therefore, desirable; and remedies such as hydrochloric acid, pepsin, pancreatin, and the like, which aid digestion, may prove beneficial. Sometimes an exclusive milk diet is useful, particularly when albuminous decomposition or putrefaction is conspicuous. In other cases, a strict limitation of carbohydrate foods is required. No general rule, however, can be laid down; as the features in individual cases vary widely.

Intestinal antiseptics of various sorts have been tried; but so far none can be said to have a generally useful effect. Small doses of calomel may be helpful, on account of their laxative as well as their antiseptic influence. Other laxatives or purgatives may be temporarily beneficial, by cleansing the intestinal tract. Strict antiseptics like betanaphthol, ichthyol, resorcin, salol, creosote, etc., have all been tried, but appear to exercise little, if any, useful effect. Sometimes, as Steele has shown, these remedies may cause more injury to the mucous membrane than to the bacteria; and may, therefore, increase, rather than decrease, the number of organisms and the activity of the putrefactive process.

Sometimes astringent remedies, such as bismuth, sulphur, and tannin, may have a useful effect; probably because they influence the intestinal mucous membrane in a beneficial manner.

Recently renewed attention has been called to the possibility of influencing intestinal fermentation and putrefaction by the administration of cultures of bacteria. An old method was to administer brewer's yeast. More recently, Brudzinski, Metchnikoff, and Tissier have employed cultures of organisms that occasion lactic acid fermentation. The same principle applies to the administration of sour milk, buttermilk, koumyss, and similar preparations. The value of this form of treatment has not yet been determined. It is doubtful whether the extravagant claims recently put forward will be substantiated by further investigation.

GASEOUS DISTENTION OF THE INTESTINES.

The terms *meteorism* and *tympanites* have been applied to the occurrence of abnormal quantities of gases in the intestines, the former signifying acute, the latter chronic distentions. Normally there is always a certain amount of gas in the bowels, consisting of the constituents of atmospheric air that have remained unabsorbed after having been swallowed, and other gases derived from the blood or from fermentative processes in the intestines. Among the gases present are oxygen, hydrogen, ammonium, carbon dioxide, methane, and sulphuretted hydrogen. The oxygen is mainly that which gains access to the bowel from atmospheric air swallowed with food. It is, however, readily absorbed, and, consequently, disappears in the small bowel, the large intestine being practically free of it (Herter). Carbon dioxide is largely derived from the blood; and the other gases are mainly derivatives of fermentative processes. The amount of gas normally present is small, and is regulated by the absorptive processes and the discharge of any excess from the stomach or bowel. Abnormal accumulations may be due to increased swallowing of air, to active gas formation within the bowel, and to interference with absorption or escape of the gas from the intestinal tract.

Increased swallowing of air sometimes occurs as a nervous disorder in hysterical persons, and has been designated by the term *aërophagia*. In some cases the patient constantly or repeatedly swallows air in a noisy manner, the swallowing being interrupted by occasional noisy eructations. Distention of the stomach first occurs; and later, some of the air passes into the bowel.

The formation of abnormal quantities of gas by fermentation is a frequent accompaniment of catarrhal conditions of the bowel and of functional intestinal indigestion. Various forms of fermentation may occur, such as those affecting principally carbohydrates; or others in which proteids or fats are involved. Sometimes such fermentative or putrefactive conditions are excited by indiscretions in diet, as when the patient eats excessively of readily fermentable food. With many individuals, milk is a common cause of the condition.

Gaseous distention due to failure of absorption or inadequate discharge of gas from the bowel is common in various intestinal diseases. Such distentions are habitually met with in cases of partial or complete intestinal obstruction. In these cases the interference with the discharge of gas from the bowel is the important factor. In catarrhal inflammations and intense congestions the accumulation of gas is due largely to the failure of absorption. This is strikingly illustrated in cases in which a portion of bowel strangulated by volvulus becomes enormously distended. The gases in such instances are partly due to fermentation within the loop, partly to the liberation of carbon dioxide from the blood of the intestinal vessels and to the stagnation of the circulation preventing reabsorption.

In peritonitis and in certain conditions of nervous or functional weakness of the intestinal walls, gaseous distention results from the inability of the bowel to remove the accumulations.

In hysterical individuals, and sometimes in persons not manifestly hysterical, extreme distention of the abdomen or localized gaseous distention may

occur. The name *phantom tumor* has been applied to this condition. In many cases it is paroxysmal and transient, but sometimes it proves quite lasting. Many of the instances, however, of supposed chronic phantom tumor have doubtless been cases of idiopathic dilatation of the colon. The explanation of phantom tumors has occasioned some difficulty. There is, probably, in most cases, an actual increase in the amount of gas; but the distention is, in-part, more apparent than real, being dependent to some extent upon contractions of the diaphragm and of the abdominal muscles, causing a greater distention of parts of the abdomen. In other cases, a paresis of the abdominal muscles doubtless plays a part. The administration of anesthetics frequently relieves phantom tumors immediately.

Symptoms.—The important symptom of meteorism, or tympanites, is the distention of the abdomen. This is usually uniform; but in some cases it affects limited portions of the bowel, and therefore of the abdomen, more than others. Localized collections of gas are not infrequent in association with organic conditions of the bowel. The degree of distention may vary from slight fullness to enormous enlargement, the amount depending upon the resistance offered by the bowel itself and by the abdominal walls. Subjective symptoms are rarely wanting. In acute cases there is often marked discomfort or severe colicky pain. In the more chronic forms constant slight abdominal distress, accompanied from time to time by exacerbations of distention, is complained of. Loss of appetite, coated tongue, nausea, and a tendency to vomiting after slight indiscretion in diet, and other evidences of disordered digestion are frequent. General symptoms, such as drowsiness, listlessness, and a variety of nervous manifestations, including headaches, neuralgias, and even pronounced neurasthenic symptoms, are common.

On physical examination, the abdomen presents a certain type of elastic resistance that is usually quite characteristic; and on percussion the deep note of tympany is more or less pathognomonic. The liver and spleen may be displaced, and their normal areas of dulness obscured or almost obliterated. This is particularly important in the case of the liver, on account of the possibility of mistaking tympany for the accumulation of gas in the peritoneal cavity.

In extreme cases the diaphragm is forced upward, and the heart may be displaced, sometimes to the extent of two or three intercostal spaces, and the bases of the lungs may be somewhat compressed. In these instances signs of pulmonary and circulatory embarrassment are usually present.

In cases of phantom tumor symptoms may be wanting, or the patient may complain of various subjective manifestations. A local gaseous distention may simulate an actual abdominal tumor. When the distention is extreme and the normal tympanitic note gives place to a duller tone on percussion, the simulation of a tumor may become quite confusing.

Diagnosis.—The diagnosis of meteorism rarely occasions difficulty. The important question in the diagnosis is to determine the cause, and this requires a careful consideration of the many conditions in which such distention may occur.

Treatment.—In cases of moderate intestinal distention due to irritative and fermentative conditions in the bowel, restriction and regulation of the diet may speedily relieve the condition. Remedies may, however, be required to reduce the distention more speedily. Various carminatives, such

as oil of peppermint, menthol, chloroform, Hoffmann's anodyne, asafoetida, and the like, are employed for this purpose. It is claimed that some of these have the effect of promoting absorption of the gas. In general, however, their effect seems to be to cause eructation or discharge from the bowel.

Sometimes the use of laxatives or purgatives may be beneficial, but usually evacuation of the bowels is best accomplished by the employment of enemata containing asafoetida or turpentine in saline solutions, soapsuds, etc. When the distention is excessive, a rectal tube may be useful in causing a rapid discharge of gas.

The peristalsis of the bowel may be stimulated in cases of muscular or nervous atony by the use of eserine. Hypodermic injections of the sulphate of eserine, in doses of gr. $\frac{1}{100}$ to $\frac{1}{50}$, repeated at intervals of three or four hours, sometimes act promptly and most satisfactorily.

Puncture of the distended bowel with a hollow needle has been practised in some cases, but is a dangerous mode of treatment and cannot be advised.

INTESTINAL INDIGESTION.

This term is preferable to the name intestinal dyspepsia, sometimes applied to it, because the latter suggests the gastric digestion rather than that of the intestines. In a broad sense, intestinal indigestion may be used as a term denoting any form of interference with the normal intestinal functions, but it is more properly restricted to functional derangements, rather than to such as may attend organic disease of the bowel. In inflammatory conditions of the intestines, in obstruction, in cases of ulceration, tumors, and the like, the normal digestive processes and absorption are interfered with; but these are properly considered as direct consequences of organic troubles. Leaving such cases out of consideration, there remains a group of conditions in which, without gross organic disease, disturbance of the intestinal functions is met with.

Etiology.—Functional intestinal indigestion may be due to (1) lack of bile and pancreatic juice; (2) lack of secretion of the intestinal ferments proper; (3) bacterial fermentative processes.

Attention is directed to the effects of the withdrawal of biliary and pancreatic secretion, in the discussion of fatty stools, acholic stools, and diarrhoea. Usually, in the consideration of intestinal indigestion, these conditions are not taken into account.

Unfortunately, very little can be said regarding the functional results of insufficient secretion of intestinal ferments proper. That these secretions play an important role in the functions of the intestinal tract is entirely probable, in view of our present knowledge; but the clinical significance of derangement of these secretions has not yet been determined with any definiteness.

Disturbance of the intestinal digestion may be a secondary result of the disorders of gastric digestion. In cases of hyperacidity of the gastric contents the alkalinity of the bile and pancreatic juice may be insufficient to neutralize the acidity of the chyme, in consequence of which the reaction of the contents of the duodenum and jejunum remains acid, instead of being alkaline. This interferes with the normal pancreatic digestion, and at the same time subjects the mucosa of the upper small intestine to an irritation that may be

sufficient to produce an actual catarrhal inflammation. A similar effect may follow insufficient secretion of bile and pancreatic juice, in which case the normal acid chyme may fail of neutralization. Much more common than either of these occurrences is the development of acidity in the small intestine as the result of bacterial invasion. This is very commonly met with in infants and children. Bacterial decomposition of milk and other foods may occasion the formation of lactic, acetic, and other acids, and may thus interfere with pancreatic digestion and, at the same time, occasion irritations of the mucosa of the small bowel.

The effects of these conditions, all of which are alike in the fact that the contents of the upper bowel are acid, are, first, an interference with normal intestinal digestion; second, an increased liability to bacterial activity; and third, irritations of the mucosa.

Symptoms.—The symptoms usually met with are partly gastric and partly intestinal. Among the gastric manifestations, nausea, vomiting, flatulence, and sensory disturbances, such as heaviness or pain, may be mentioned. Among the intestinal manifestations are gaseous distention and diarrhoea. The acid contents provoke an irritation of the upper bowel and the formation of excessive quantities of mucus. At the same time, peristalsis is increased by the irritation; and the semifluid contents of the upper bowel are hurried into the lower bowel and discharged before normal inspissation and chemical changes have time to occur. The stools, therefore, are acid, instead of alkaline, and frequently cause marked irritation of the rectum and anus. In children this becomes an important symptom; and intense irritation, sometimes extending to a considerable distance over the buttocks, may be met with. The movements are usually somewhat tenacious, on account of the admixture of excessive quantities of mucus, and commonly present a greenish color, on account of the presence of bacterial pigments or of unaltered bile. In a special form described by Schmidt, under the name of *intestinal fermentative dyspepsia*, the stools are of very light color, foamy, and of a musty or sour odor, due to the presence of butyric acid.

When functional intestinal indigestion persists, the irritation of the bowel readily passes into a condition of catarrhal inflammation.

Diagnosis.—The diagnosis of intestinal indigestion often presents great difficulty. In some instances the symptoms are vague and indistinct; and it may be difficult to determine that a continuous tendency to intestinal flatulence and slight disorder of the bowel is due to a purely functional disturbance rather than to organic disease. In more acute cases, especially those occurring in children, the evidence is more trustworthy. The occurrence of mucous, gelatinous stools of green color, acid reaction, and sour or musty odor, is strongly significant of the form of disease now under consideration. It is always difficult, however, to determine that an actual catarrhal enteritis has not supervened.

Treatment.—The treatment of this condition requires close supervision of the diet. In the beginning of acute attacks, the withdrawal of all food may be advisable. In children, as well as in adults, the substitution of albumin water, broths, soups, or barley water for the ordinary diet, and especially for a milk diet, is advisable. Later, the food should be regulated according to the nature of the case. Sometimes, when fermentative processes and gas-formation are conspicuous, it is best to use only animal

food, such as eggs, soups, jellies, and gelatin preparations; while in other cases, in which albumin decomposition predominates, gruels, cereals, and farinaceous foods generally, with smaller amounts of albuminous food, should be given.

The medical treatment is directed to the evacuation of the contents of the intestines and the removal of fermenting material. A full dose of castor oil, or repeated small doses, may accomplish this purpose. Calomel in small amounts (gr. $\frac{1}{40}$ to gr. $\frac{1}{10}$), two or three times daily, may exercise an antiseptic effect, as well as a slightly laxative influence. Other remedies like salol, betanaphthol, guaiacol carbonate, and ichthyol may be useful as antiseptics. After the upper bowel has been freed of fermenting contents, bismuth, chalk mixture, or other mild astringents may be desirable to allay any existing irritation and check the tendency to further diarrhœa. Pancreon, prepared by treating pancreatin with tannin, is recommended when intestinal indigestion is due to decreased secretion of pancreatic juice. It is administered dissolved in water before meals in a dose of gr. vj (gm. 0.4.).

In cases in which gastric disorders have preceded the intestinal disturbance the treatment should be directed primarily to the condition of the stomach. Further discussion of the treatment of bacterial disturbances of the bowels will be found in the section on Bacteriology of the Intestinal Tract.

INTESTINAL COLIC.

Definition.—The term intestinal colic should be restricted to attacks of paroxysmal intestinal pain caused by violent tonic contractions of the bowel resulting from undue stimulation of the intestinal nerves or from obstructive conditions. The more or less continuous pain and tenderness of inflammation either of the bowels or peritoneum, and the purely neuralgic pain (*enteralgia*, in the strict sense) occasionally met with in persons suffering from organic or functional nervous diseases should not be included under the head of colic.

Etiology.—The immediate cause of the pain of colic is the excitation of the intestinal nerves and the violent tonic contraction of the bowels caused by irritating intestinal contents or by obstructive conditions. The cases may be divided into those in which there is mainly functional disturbance and those associated with organic conditions.

Colic Due to Functional Disturbances.—Some persons are so sensitive that even when the food or other ingesta are wholesome, slight causes such as exposure to cold, fatigue, anger, fright, or other emotional conditions occasion attacks of colic. It is probable that under these circumstances digestion and absorption of food products are interfered with, fermentation or putrefaction occurs, and irritation of the intestinal nervous mechanism takes place. More commonly, colic results from a definite error in diet. The eating of coarse foods, unripe fruit, indigestible substances of all sorts, especially if in excessive quantity, is perhaps the commonest cause. The drinking of cold water, beer, or other liquids at the same time may add to the disturbance, or may alone suffice to bring on an attack. Developmental conditions causing functional weakness or chronic conditions of the bowel, such as partial stenosis, may predispose to attacks of colic under relatively slight provocation, such as overeating. In infants or young children the connection between

dietetic errors and colic is nearly always apparent. Frequently such co-operating causes as exposure to cold, fatigue, or emotional disturbances may play an important etiological part. Idiosyncrasy also contributes, some persons finding it impossible to eat certain, usually wholesome, articles of food without suffering an attack. Whether the ingested food is inherently irritating or is rendered so by the conditions of the patient at the time, or his idiosyncrasies, the result is a violent irritation of the nerves of the intestines and a forcible contraction of the muscles of the intestines which causes the painful paroxysms. If the amount of food residue and gaseous accumulations in the intestinal canal is excessive, the obstacle thereby offered to the contractions of the bowel becomes an added cause of pain. Nearly always some sort of obstacle to the free movement of intestinal contents is more or less responsible for attacks of colic. In some cases violent irritation of highly noxious substances may occasion a spasm or paresis of a segment of bowel which thus becomes practically a seat of obstruction. Mere distention of the bowel by gases does not occasion colicky pain, as may be seen in the comparative freedom from all pain in cases of quite marked meteorism and the different type of discomfort or pain in cases of excessive distention.

Certain special substances may act in a direct way upon the nervous mechanism of the bowel. This is seen in the intense colic of lead poisoning and the colicky pains caused by certain laxative medicines. Occasionally infectious diseases, such as influenza and malaria, begin with an attack of colic, which may perhaps be due to disturbance of digestion at the onset of the disease or to some special effect of the disease itself.

Formerly much was said about rheumatic colic. The cases so designated were cases of colic following exposure to cold and associated with more or less muscular soreness. Some of these doubtless were simply instances of painful conditions in the abdominal walls; others cases of colic due to intestinal indigestion, fermentation, etc., following the eating of indigestible food, and exposure. It is doubtful if any of the cases were instances of "rheumatic colic" in a proper sense.

Fæcal accumulations may cause purely functional colic, although they are usually associated with some organic condition. When a considerable impaction has occurred, the peristaltic efforts of the bowel above the accumulation become more and more severe until finally tonic contraction ensues and an attack of colic results. Neglect in the regular evacuation of the bowels and overfeeding are common causes of this form of colic in some persons.

Colic Due to Organic Conditions.—Foods may prove so irritating to the bowel that an actual catarrhal inflammation is occasioned. This happens quite frequently in children when tainted milk is taken and in adults when bacterial decomposition causes putrefactive changes. The catarrhal inflammation adds to the irritability of the intestinal nerves and increases the tendency to an attack of colic. Ulceration of various sorts of the large or small intestine, such as tuberculous, typhoidal, stercoral, or simple follicular, may occasion genuine attacks of colic. The association of colic with appendicitis is exceedingly common, and may be due to violent contractions of the appendix itself (appendicular colic) or of the ileum or colon. All these inflammatory lesions may excite tonic peristaltic contractions, and the partial obstruction at the site of inflammation increases the obstacle to the movement of the intestinal contents from above. The association of attacks of

colic with various forms of acute and chronic intestinal obstruction will be considered in the discussion of that condition.

Symptoms.—The ordinary attack of intestinal colic due to functional disorder of the bowels is usually sudden in onset, beginning with violent cramp-like pains in the umbilical region which spread in various directions. As the peristaltic movements advance from one part to another the patient experiences a change in the position of the pain, and there may be at the same time a sense of movement of gases from one coil of intestines to another. In very severe attacks the pain is more fixed and cannot be definitely localized. The patient at first moves from side to side, draws up his leg, and presses his hands or a pillow against the abdomen, and finally assumes a constant position with thighs flexed toward the abdomen. Pressure usually affords relief, but if inflammatory conditions of the bowel are present, pressure may increase the pain. In young children an attack of colic is indicated by crying, restlessness, drawing up of the legs, violent contractions of the facial muscles, and later by abdominal distention. Rumbling sounds or borborygmi may indicate the movement of gases in the bowels. Usually attacks of colic subside after a duration of from a few minutes to some hours, and when gas has been discharged or the bowels evacuated. In severe cases the attacks may continue unabated for several or many hours and the patient may grow faint or suffer partial collapse, breaking out into a cold sweat and growing quiet from exhaustion. Severe attacks may also be attended with nausea and vomiting or repeated retching. The temperature in functional colic is usually little altered, but a moderate degree of fever is not uncommon.

After relief from the primary attack, exacerbations and remissions may follow until the bowels have been thoroughly evacuated or the irritation otherwise removed. A certain amount of abdominal soreness and tenderness may remain after the attacks have ceased and a return to normal intestinal conditions may be delayed for several days.

The bowels at the onset of colic are usually constipated. When relief begins there is usually some discharge of flatus and evacuation of the lower bowel; later, a diarrhoeal condition may follow, particularly if catarrhal inflammation has accompanied the attack or occasioned it.

Attacks of colic in which the irritation has been sufficient to cause an inflammatory condition are more severe and lasting than those in which the derangement is only functional. Disturbances of the stomach, abdominal distention, soreness and fever are also more likely to occur in these cases; and after relief from the acute condition tenderness and pain, diarrhoea, and sensitiveness of the bowels are apt to persist for some time. The symptoms of such cases will be more appropriately described under the head of catarrhal inflammation of the bowels.

Colic, associated with organic diseases of the bowels, such as intestinal obstruction from carcinoma, bands, internal hernia, faecal impaction, etc., will be considered in the discussion of those conditions.

Enteralgia or *neuralgia of the intestines* in the strict sense is a rare condition, and to be distinguished from colic by reason of the fact that it is probably not dependent on a tonic intestinal contraction, but rather on a direct painful irritation of the intestinal nerves. Such a condition occurs in hysterical persons and in association with tabes and other organic spinal diseases. The attacks are sudden and severely painful, but unattended with the usual indications of colic. There is intense sensitiveness on pressure, and

relief occurs independently of evacuation of the bowels. The general appearance of the patient suggesting a neurotic origin of the pain is more or less characteristic.

Diagnosis.—It is often exceedingly difficult to determine the nature of an attack of abdominal pain having the general features of intestinal colic. The serious character of other conditions which simulate its features requires that these be carefully considered before an attack is regarded as simply intestinal colic. Among other conditions, biliary, renal, and uterine colic must be thought of; the similarity in the symptomatology of rupture of a gastric or duodenal ulcer must be remembered; and above all else, on account of its frequency and seriousness, appendicitis must be kept in mind. The last-named offers the greatest difficulty; but if local pain, tenderness and rigidity, fever, leukocytosis, marked gastric symptoms, obstinate constipation, disturbances of the pulse, and the general condition of the patient be carefully considered, a mistake in diagnosis is not likely to be frequent.

Certain less frequent conditions may require consideration. Among these, acute pancreatitis, the passage of a pancreatic calculus, embolism or thrombosis of the mesenteric vessels, and referred pains, such as those occasionally experienced in the abdomen in cases of thoracic diseases, as pneumonia, pleurisy or angina pectoris, or in pelvic diseases, must be remembered. The onset of acute intestinal obstruction must be carefully distinguished. Usually the failure to relieve the attack by simple measures establishes the diagnosis.

In cases of chronic intestinal obstruction the significance of attacks of colic may be overlooked. A partial obstruction may be unattended with marked symptoms until an attack of acute colic occurs. The possible association of this with a chronic condition may be suggested by the age and general physical conditions; at all events, the possibility of a merely symptomatic colic must be remembered.

Treatment.—Intestinal colic readily subsides when the bowels are evacuated and the local irritation is controlled by various warm applications and by the use of sedatives. If the diagnosis is assured, the bowels may be emptied by the use of a large injection of warm water or soapsuds, to which castor oil or sweet oil may be added; or by the administration of a dose of castor oil if the stomach permits. Salines, calomel, or other purges may be used if the urgency of symptoms does not require speedier agents.

Small doses of opium or morphine not only allay pain, but relax spasm, and thus sometimes aid in securing purgation. Various carminatives, such as spirits of chloroform, oil of peppermint, menthol, or camphor water may be combined with opiates, especially when gaseous fermentation is present. A prompt hypodermic injection of morphine followed by measures to relieve the bowels may cut short an attack of threatening severity.

Local measures, such as hot-water bags, hot fomentations, poultices, etc., give relief, and by relaxing spasm may secure evacuation of the bowels.

In children, hot applications applied locally, hot baths, carminatives with paregoric, mild laxatives, such as castor oil with paregoric, magnesia, calomel, and enemata suffice for severe attacks, while the milder paroxysms are relieved by using soda mint, oil of peppermint, aromatic spirit of ammonia, or Hoffmann's anodyne.

LEAD COLIC.

A peculiar form of colic is that which is met with in subacute lead poisoning, and commonly known as lead colic, painter's colic, etc. In this form, the important symptom is intense cramp-like pain attended with obstinate constipation. The abdominal muscles are found rigid and contracted, so that the abdomen is flat or even hollow. Other indications of lead poisoning are commonly present, such as ashy pallor and more or less decided anæmia, in which a pronounced reduction in the percentage of hæmoglobin and the occurrence of marked changes in the red corpuscles (such as loss of color, polychromatophilia, pigmentary degeneration, and nucleation) are conspicuous. A blue line of discoloration along the edges of the gums where they join the teeth is almost pathognomonic. Paralysis of the extensor muscles of the forearm, causing the characteristic wrist-drop, may or may not be present. It rarely attends the early attacks of colic, but is quite usual in recurrent paroxysms. During the attack of colic, the pulse has a peculiar wiry hardness, indicating a high tension, which has been ascribed to tonic contraction of the bowels and consequent increased resistance to the abdominal circulation. These symptoms, in conjunction with the history of occupation or habits, usually suffice to establish an accurate diagnosis.

Treatment.—The treatment of lead colic is directed to the relief of the symptoms and the elimination of lead from the system. In the early stages magnesium sulphate should be given freely for the double purpose of converting any soluble lead salts in the stomach or bowels into the insoluble sulphate and of relieving constipation. At the same time opium may be given by the mouth or rectum, or hypodermics of morphine may be used to allay pain and relieve intestinal cramps and spasm. Free venesection gives prompt relief in some cases. Iodide of potash in doses of from ten to fifteen grains (gm. 0.65 to 1) three times daily aids in eliminating lead from the tissues.

INTESTINAL HEMORRHAGE.

The terms intestinal hemorrhage and enterorrhagia are sometimes used in contradistinction to the term *melæna*, the former being intended to convey the idea of mere hemorrhage from the bowel, while the latter implies a hemorrhagic diarrhœa. It is difficult, however, to maintain this distinction practically, because hemorrhages from the bowel and hemorrhagic diarrhœas merge into each other without any sharp line of division.

Etiology.—The causes of hemorrhage from the bowel are exceedingly numerous. In some cases this condition occurs without any definite organic lesion of the intestines; but more commonly, hemorrhage signifies some definite change.

Hemorrhage Unassociated with Pathological Conditions of the Bowel.—In association with certain hemorrhagic diatheses or diseases, such as purpura and scurvy, and in some infections, such as septicopyæmia, yellow fever, and severe malaria, hemorrhage may occur without preceding alteration in the mucous membrane of the bowel. At the time of the hemorrhage, or before it sets in, extravasation of blood into the mucosa may occur; and the source of the hemorrhage may be such an area. Occasionally hemorrhages

occur from the mucous membrane of the bowel without leaving any trace of their source, and, therefore, without any discoverable anatomical lesion. Cases of this kind have been met with in tuberculous individuals by Nothnagel and others. It has been claimed that hemorrhages of this sort may take place as a vicarious manifestation of menstruation. This occurrence is probably, however, rather rare.

Hemorrhage as the Result of Congestive Conditions of the Bowel.—In cases of cirrhosis of the liver, valvular disease of the heart, and hemorrhagic infarction of the bowel following embolism or thrombosis of the mesenteric vessels, hemorrhage may result as the consequence of the engorgement of the intestinal circulation. Similarly, in cases of intussusception, strangulation of the bowel, or hernia, there may be hemorrhage as the result of overdistention of the bloodvessels.

Hemorrhage as the Result of Diseases of the Bloodvessels.—The most frequent cause of hemorrhage from the bowels is hemorrhoids. Other diseased conditions of the bloodvessels may, however, occasion hemorrhage. Thus in aneurismal dilatations of the hepatic artery, rupture may take place into the bile passages; and discharge of blood from the bowel may ensue. Similar conditions of the intestinal vessels occur more rarely. Amyloid degeneration of the intestinal vessels is a common cause of extravasation into the bowel in local or general amyloid disease. In grave anæmias and in severe infections, degeneration and rupture of the bloodvessels may be the cause of enterorrhagia. Sometimes in the course of chronic interstitial nephritis considerable intestinal hemorrhage may be met with, probably resulting from degeneration of the bloodvessels and increase of arterial tension.

Hemorrhage as the Result of Inflammatory and Ulcerative Conditions of the Bowel.—In cases of gastric and duodenal ulceration and in typhoid fever hemorrhage occurs frequently. Less marked hemorrhage often attends carcinomatous ulceration of the bowel; while in ulcers of other sorts, loss of blood, although less marked and conspicuous, may be met with.

In inflammatory affections of the bowel without ulceration, hemorrhage is much less commonly present. If, however, the inflammation is intense and the congestion is marked, some loss of blood may occur, although free hemorrhage is unusual. Continued slight bleeding is caused by certain parasites, notably ankylostoma. High grades of anæmia may result from this cause.

It is important to recognize that in cases of gastric ulcer hemorrhage from the intestines sometimes occurs without any hæmatemesis; and that the loss from the bowel is much more pronounced in some of the cases in which hemorrhage from the stomach also occurs.

Symptoms.—Ordinarily, no difficulty occurs in the recognition of hemorrhage from the bowels. It may, however, be difficult to determine the source of the hemorrhage. As a rule, when the blood comes from the colon or rectum, its color is a bright red, and on microscopic examination the corpuscles are well preserved. When hemorrhage occurs from the upper bowel, unless active peristalsis causes its rapid discharge, secondary changes are usually observed. The stools become dark and often tarry in appearance, and are usually highly offensive from decomposition. In many cases there may be a gradual loss of blood from the bowel of so slight a degree that a naked-eye inspection of the stools does not reveal any evidence of hemorrhage. In such cases an examination for occult blood may show its presence.

In cases of larger hemorrhage a microscopic examination may discover crystals of blood-coloring matter and dark amorphous pigment.

In cases in which inflammatory lesions, ulceration, and especially carcinoma, have occasioned hemorrhage from the bowels, the blood is often associated with mucus and pus, as well as with epithelial cells and other tissue elements.

Hemorrhage from the bowels rarely occasions pain. If the loss of blood is great, the symptoms are those of extensive hemorrhage from any source. After a hemorrhage, intestinal symptoms often develop as the result of putrefactive changes in the blood retained in the intestinal tract. Secondary diarrhoea may be set up as a consequence.

Repeated small hemorrhages from the bowel, such as occur in cases of extensive ulceration, in bleeding piles, or when blood-sucking parasites, such as ankylostoma, infest the intestinal tract, may occasion extreme grades of anæmia, which are with difficulty distinguished from progressive pernicious anæmia. In most cases the blood picture is that of the secondary anæmias; but in some the resemblance to pernicious anæmia is extremely puzzling.

Prognosis.—The result of intestinal hemorrhage depends entirely upon its cause. Severe losses of blood are, of course, in all cases serious; but sometimes very extensive hemorrhages are followed by no material impairment of the patient's health. In cases of hemorrhoids, as well as in cirrhosis of the liver or other obstructions of the portal circulation, actual improvement may be observed. Repeated losses of blood are of more moment; and when intense anæmia has developed, the prognosis is always very grave.

Treatment.—The treatment of intestinal hemorrhage must always depend upon the cause, although certain general rules may be formulated that are applicable to nearly all cases.

Local applications of cold, such as the ice-bag, Leiter's coils, and the like, are commonly employed, although their value is extremely doubtful. Injections of ice water into the rectum and colon may, perhaps, be valuable in some cases of hemorrhage from these parts, but in other instances may be actually harmful, by stimulating the peristalsis of the bowel.

Opium has been universally employed for its effect in controlling peristalsis and quieting the patient. The judicious use of this drug is advisable in some forms of intestinal hemorrhage; but it should not be used to such an extent that the bowels are completely locked and the blood and other materials therefore retained and permitted to undergo putrefactive decomposition. Astringent remedies, such as nitrate of silver, acetate of lead, bismuth salts, and tannic acid preparations, are sometimes useful. Other hæmostatics, like chloride or lactate of calcium and gelatin, may be used. In the case of the latter, peristalsis should not be checked, so that the gelatin may reach the bowel promptly and in an undigested form. The products of gelatin digestion exercise no local hæmostatic effect—perhaps even the contrary, while the unaltered substance appears to have some influence in controlling hemorrhage when locally applied.

Ergot, hydrastis, and turpentine have been used in the treatment of intestinal hemorrhages, but are of doubtful value.

Colonic douches may be beneficial when the source of the hemorrhage can be reached in this way. Solutions of nitrate of silver, tannic acid, or

other astringents, may be employed. If the hemorrhage cannot be reached, such injections, however, may be objectionable; because, by irritation, they may stimulate active peristalsis and renewed hemorrhage.

CONSTIPATION.

Synonyms.—Sluggishness of the bowels; obstipation.

Definition.—Infrequency in the evacuation of the bowels and a tendency to abnormal dryness or hardness and reduction in the quantity of the feces are the conditions comprised in the term constipation.

It is difficult to give a positive definition, since the physiological limitations vary widely. In most healthy persons there is normally an action of the bowels once in twenty-four hours, and when the conditions of life are uniform this occurs at approximately the same time of day. When the intervals are two or three days, even though symptoms are absent, the condition must be regarded as one of constipation. Instances, however, have been reported, such as the cases of Reichmann, in which there was but one movement in two or three weeks, the patients in the meantime presenting no symptoms of disease. Such cases must be regarded as cases of decided constipation. Sometimes, although the movements occur daily, the quantity is decidedly below normal and the character of the movements inspissated. In other cases, by testing with charcoal or carmine, it may be found that, although an approximately normal movement is evacuated daily, the duration of time in the intestinal tract has been prolonged, so that considerably more time elapses between the ingestion of food and the discharge of the residue than occurs in normal individuals. This condition has been defined as latent constipation.

Essentially, then, delay in the evacuation of food residues and abnormal inspissation, with a reduction in the quantity of the excreta, constitute the condition under discussion, whether symptoms be present or not.

Etiology.—A recognition of the causes of constipation requires a knowledge of the normal mechanism of intestinal movements and of the final evacuation of the fecal residues. Two forms of movement have been recognized in the intestines by direct observation in man or animals when the abdominal cavity has been laid open, and by studies made with the aid of the x-rays of the movements of the intestinal contents containing non-permeable substances, as bismuth subnitrate. First, there may be recognized swaying movements (Raiser) or rhythmic segmentation (Cannon), which consist of contractions and relaxations of segments of the bowel parallel to its axis. These movements occupy but a few centimeters of the length of the bowel, and serve the purpose of dividing the contents of the bowel into short segments and of thoroughly commingling the contents with the intestinal secretions. The second form of movement or peristalsis is an alternate dilatation and contraction of the lumen of the intestinal lining as a wave of moderate rapidity which extends considerable distances along the bowel. There is, as Starling observes, a contraction behind some object and relaxation in front, so that the contents are moved onward by the contraction. The peristaltic wave does not run the full length of the bowel, but extends a certain distance and then ceases, to be renewed again and carried farther.

The movements of the intestine are caused by mechanical and chemical

irritations acting upon the inner lining of the bowel. Solid contents, water and other liquids, or gases, may serve as stimulants either mechanically or chemically. Sometimes the irritation results from the mere bulk of the contents; more commonly, the mechanical nature of the contents determines their effect. Thus, coarse and irritating substances and insoluble residues of various sorts are particularly exciting. Of gases, hydrogen, oxygen, and nitrogen are said to be indifferent, while carbon dioxide, sulphuretted hydrogen, and volatile hydrocarbons stimulate activity.

The nervous stimuli proceed from the plexuses of Auerbach and Meissner. Some difference of opinion has arisen regarding the manner of transmission of the impulses from the upper to the lower bowel. Englemann has claimed that the stimulus travels through the muscle cells, while Nothnagel and others assert that nervous paths are always necessary. The movements of the small intestine are undoubtedly under direct nervous regulation, the impulses travelling from the spinal cord through the sympathetic plexuses. Motor impulses reach the intestine through the vagus, and irritation of this causes movements of the small intestine and the upper half of the large. Irritation of the splanchnic nerve causes inhibition of movements, but some recent observers have insisted that the splanchnic also contains motor fibers for the longitudinal muscles. Others deny any motor function to the splanchnic. Cannon recently has shown that section of the splanchnic or of the vagus and splanchnic disturbs peristalsis very little. The lower half of the large intestine is innervated through the inferior mesenteric plexus and hypogastric plexus.

It is also of importance that the splanchnic nerve contains vasomotor fibers which cause contraction of the intestinal vessels and thus indirectly influence both secretion and the movements of the bowels.

The onward movement and eventual evacuation of the residual matters left in the bowels after complete digestion is the result of the peristaltic movements above described. Through this agency the contents traverse the small intestine in the course of a few hours and the large intestine in from twenty to twenty-four hours. The slower progress in the colon is probably the result of the frequency of antiperistaltic waves, which Cannon has found to be the prevalent form of motion in this portion of the intestinal canal. As a consequence of this delay the absorption of water from the intestinal contents takes place mainly in the colon, and the fæces undergo a gradually increasing solidification.

The final evacuation of the fæces is caused by reflex stimuli produced by irritations in the rectum resulting from the presence of fæces and the direct voluntary expulsive efforts which involve contractions of the abdominal muscles.

The condition of the abdominal muscles plays a further part in the whole process of the movement of the intestinal contents in that the condition of these muscles and the effect of their normal contraction influences very largely the activity of the peristaltic contractions of the intestines.

It will be seen then that abnormal sluggishness in the onward movement of the contents of the bowels may be the result of (1) the character of the food residues; (2) weakness of the intestinal muscles; (3) weakness of the nervous mechanism involved in peristalsis; (4) weakness of the abdominal walls. In addition to these causes, which operate in a functional manner, there are certain organic conditions which directly oppose the onward

movement of the bowel contents. The causes of constipation then may be considered as being functional and organic.

Functional Causes.—There may be considered under this head conditions which produce torpor of the intestines themselves and conditions which cause a weakness of the diaphragm and abdominal walls, and thus lead to reduction in the expulsive power or indirectly to weakening of the intestines themselves.

Intestinal torpor may occur acutely and as a result of various nervous conditions. Thus, as a result of fear or other emotions and nervous manifestations in neurasthenia, melancholia, and the like, intestinal atony and constipation are not infrequent. Frequently this torpor results from habit. The normal individual tends to evacuate the bowel after the first meal of the day, probably for the reason that at this time the food residue of the previous day has reached the lower bowel and that the ingestion of food into the stomach, together with the change of position of a person from the recumbent to the upright, and the exercise taken up with the day's work, excites peristaltic contractions and the tendency to evacuation. When, as a result of lack of time or inattention, this stimulus is ignored or resisted, the desire passes, and with repetition of such neglect, the habit of longer retention is established. The normal stimulus from the lower bowel becomes less and less effective, and eventually a state of chronic constipation results. Long retention of fecal matter in the lower bowel has the further unfavorable effect of causing dilatation of this portion of the bowel and an active organic weakening of its expulsive power. In individuals suffering from long-continued constipation, considerable pouching of the sigmoid, descending colon, and rectum is not infrequent.

Diseases of the stomach frequently occasion chronic constipation. In some instances this may be the result of the long retention of food in the stomach owing to the reduction of its motor power or obstruction of the pylorus, and the stimuli which are propagated from the stomach to the intestine fail of their normal effectiveness. In addition, the contents of the stomach may be transmitted to the intestine in such condition that irritation of the bowel is first produced, and later spasmodic or tonic contractions cause the resulting constipation.

The dietetic causes of constipation are numerous and most important. The habitual use of food which leaves little residue is a very frequent cause. The constipating effect of a milk diet is thus explained. Similarly, the use of highly concentrated food, such as meat, eggs, etc., with little addition of such substances as vegetables, which contain a relatively large amount of unabsorbable residue, may produce habitual sluggishness of the bowels. The neglect of vegetables, besides causing a reduction in the amount of residue, has the disadvantage of removing from the dietary a large proportion of the neutral salts which are normally active in regulating the action of the bowels. Occasionally the bowels become constipated as a result of the eating of foods which contain an excess of irritating residue, such as berries containing small seeds or vegetables rich in cellulose. This form of constipation is due to spasmodic conditions of the bowel caused by the excessive irritation. Some individuals are more susceptible to such conditions than others.

In many persons the cause of constipation appears to be insufficient use of water, which leads to a too great inspissation of the intestinal contents. On the other hand, the drinking of hard water containing large quantities of lime salts may be at fault.

Bacteria play an important role in the normal as well as in the abnormal conditions affecting the intestinal tract. Recent observations have shown that the number of bacteria in the faeces of constipated persons is decidedly smaller than in those of normal individuals. This is, in part, due to the fact that the sluggish peristalsis occasions a longer retention of food in the upper bowel, and consequently a fuller degree of absorption of the nutritious contents. As a result, the bacteria which mainly flourish in the lower intestinal tract are deprived of pabulum and multiply less freely, in consequence of which, fatty acids, gases, and other products of bacterial growth are wanting and the stimulus to peristalsis normally exercised by these products is lacking. To some extent the bacteria also excite mucus formation in the normal bowel, and this aids as a lubricant and helps to keep the faeces soft.

Congestion and inflammation of the bowels in certain cases cause constipation by directly weakening the contractions, but this mode of causation of constipation is probably far less frequent and important than has commonly been assumed. As a matter of fact, it would appear that chronic constipation is more usually the result of disorder of the nervous mechanism which controls peristalsis than of any direct weakness of the wall of the bowel. It cannot be denied that inflammations or congestion, and especially such conditions as congenital or acquired dilatation of the bowel, may produce constipation by direct weakness of the expulsive movements, and it is contended only that such factors are of relatively little importance compared with reduced motility through alterations in the contents of the bowel or of the neuromuscular mechanism that controls the peristaltic motions.

Various causes operate to produce constipation by causing weakness of the diaphragm or the abdominal walls. A direct effect is probably exerted upon the movements of the intestine by the contractions of the abdominal muscles, and, in addition, it is probable that a normal condition of tone in the abdominal walls has a certain reflex influence in producing a like state in the underlying organs. Furthermore, relaxation of the walls of the abdominal cavity allows of dilatation of the contained hollow viscera, which interferes with the effectiveness of the contraction of these organs. Clinically, it is certain that various conditions, which cause weakness either of the diaphragmatic movements or of the abdominal parietes, occasion sluggishness of the bowels. Thus, in chronic diseases of the chest, such as emphysema, pleural adhesion, etc., more or less obstinate constipation may occur. The effect of weakness of the abdominal walls is evident in the constipation that occurs after repeated pregnancies or when large accumulations of fat have been deposited in the omentum or abdominal walls, or in the case of various abdominal tumors. Intestinal sluggishness may reach high grades in cases of decided separation of the recti as a result not only of the weakness of the superficial structures, but also of displacement of the intestines (enteroptosis).

Dress probably has a certain amount of influence as a cause of constipation. The much greater frequency of this disorder in women than in men may be attributed to lack of equal exercise, to the more frequent neglect owing to inconvenience of surroundings, etc., and possibly to certain anatomical considerations; but it is not improbable that the character of clothing has something to do with this greater prevalence of sluggishness in women. The weakness of the abdominal walls which results from constriction of the lower chest and abdomen may quite readily serve as a contributing cause.

Sedentary life and lack of exercise operate in a variety of ways. In the first place, there is a tendency to digestive disturbance and a lack of general systemic tone, and in a more direct way these conditions may operate by producing weakness of the abdominal muscles. It is a matter of every-day experience that individuals normally perfectly regular at once become constipated when confined to the house or room by some trivial ailment which merely restricts the usual exercise. The same result also occurs to persons on long railway journeys or sea voyages and the like, and is especially marked in patients confined to bed through some injury or a disease not necessarily affecting the abdominal walls directly. On the other hand, one sees restoration of the normal function after temporary sluggishness when the accustomed amount of exercise is resumed.

Organic Causes.—More attention was formerly paid to the importance of strictly organic conditions in the development of habitual constipation than is now given to such conditions. Undoubtedly adhesions or partial obstructions by stenoses of localized segments of the bowel or constricting bands surrounding a coil of intestine may be the occasion of intense and long-continued constipation. Similarly, uterine displacement, pelvic tumors, malpositions of the intestine (enteroptosis), or congenital or acquired malformations (idiopathic dilatation of the colon) may be of importance in certain instances. Likewise, certain conditions affecting the efficiency of the expulsive efforts, such as a weakened perineum from lacerations, and diseased conditions or relaxation of the abdominal walls, may be of some influence. But taking all cases of habitual constipation into consideration, it will be found that such organic conditions are of comparatively infrequent occurrence, and that, as a rule, some one or some group of conditions influencing the functional activity of the bowel is the important operative agency.

Pathology.—In a majority of cases but little structural alteration can be detected, even in cases of constipation that have lasted for long periods of time. Certain resulting conditions, however, are met in a certain number of the cases. Nearly always there is some abnormal distention of the sigmoid and rectum, and when the degree of constipation has been extreme and its duration prolonged, distinct pouches or diverticula may be produced. In some instances these will be found to contain hardened scybala and inflammatory materials, and even ulceration of the mucous membrane may occur (stercoral ulceration). Hardened scybala may produce catarrhal inflammations of the mucous membrane even without the presence of diverticula. Severe forms of catarrh of the mucosa sometimes result from faecal impaction.

Marked distention of the colon, with thinning of the mucous membrane and of all the walls, may result from obstinate constipation, especially when accompanied by frequent impactions in the lower bowel. The small intestine rarely shares in such distentions. More commonly there may here be found narrowing of the lumen and thickening of the wall in limited segments due to long-continued or repeated spasmodic contractions, especially in cases in which habitual insufficiency of food has been a principal cause of the disorder. The writer has repeatedly met with such contracted areas of the bowel in autopsies in cases of chronic insanity in which there has been long-continued insufficiency of food and resulting constipation. The large intestine may be similarly affected, although perhaps less commonly.

Sometimes infiltration of the inspissated faecal masses or scybala with salts of lime or magnesium may result in the formation of so-called "coproliths." These are always found in the large intestine.

Symptoms.—The important symptom of constipation is the infrequency of evacuation or the inspissated character and reduced amount of faeces. There is usually a tendency to the formation of hardened masses or scybala. The act of evacuation is attended with difficulty and not rarely with a certain amount of local pain. When the condition is long continued and the masses are of unusual hardness, some mucous coating and even blood-streaked mucus may be observed. If the lower bowel has become impacted, cramps or colicky pains may, for some time, precede the evacuation. Not rarely a certain amount of tenderness is found in the left iliac fossa over the sigmoid flexure and is the result of irritation caused by the retained scybala.

Various subjective symptoms are met with in persons suffering from habitual constipation, and it is at times difficult to determine how far these have resulted from the condition, or, on the other hand, the degree in which they may have acted as causes of the sluggishness of the bowel. Among such symptoms, heaviness, sleeplessness, a tendency to vertigo, headaches, and even more severe nervous symptoms, like hypochondriasis, may be mentioned. Undoubtedly all of these may be occasioned by the existence of obstinate constipation, but, on the other hand, sluggishness of the bowels is frequently the result of such nervous conditions and perhaps more remotely of the gastric disorders that have occasioned these symptoms. Some authors have suggested that neurasthenia is frequently caused by atonic conditions of the bowel, with more or less constipation, but certainly more commonly constipation is a symptom of the neurasthenia than the reverse.

Local abdominal symptoms are common and significant. A feeling of fulness or of pressure, a sense of distention, and sometimes vague or acute pains may be complained of. Pain may be localized, as has already been stated, to regions where accumulated masses of hardened faeces have caused irritation or inflammatory conditions. In other instances cramp-like colic affecting the region of the descending colon or the entire abdomen may result from impaction and from the resulting stimulation of violent peristalsis. Rectal impactions occasion sensations of bearing-down or perineal fulness and even violent tenesmus.

Secondary disorders of digestion, with loss of appetite, coating of the tongue, sleepiness, thirst, and sometimes more acute symptoms, such as nausea and vomiting, may, from time to time, make their appearance or may be more or less constant.

Fever not infrequently occurs from time to time, when the degree of constipation is temporarily more marked than usual. This may be the result of the direct irritation of retained masses of hardened material or may possibly be the consequence of the absorption of products of decomposition (stercoræmia). The evidence in favor of the latter is rather insufficient, as the indications of intestinal fermentation or putrefaction are rarely marked in constipation, and other signs of toxæmia are wanting. In the great majority of cases of constipation fever does not occur.

Local disorders of various sorts may be encountered, such as palpitations of the heart, irregularity of cardiac action, and various nervous symptoms, some of which have been already mentioned. These symptoms, like fever

and cerebral manifestations, have been attributed to auto-intoxication, although the evidence in favor of this view is scanty and uncertain.

The abdomen is sometimes flat or even scaphoid from general contraction of the intestines and insufficiency in the contents of the bowel. More frequently there is slight distention with excess of tympany in the sides due to dilatation of the large bowel. When the abdominal walls are weak and thin, the dilated colon may be seen almost throughout its entire course.

Hardened faecal masses may be palpable through the abdominal walls, especially in the sigmoid region. The character of the masses is recognized by their movability, by their disappearance after purgation or the use of enemata, and, according to Gersuny, pressure upon the faecal tumor causes a characteristic sensation of separation of the wall of the bowel from the tumor when the pressure is relaxed. Sometimes faecal accumulations occur in the transverse colon or even in the caecum, and may be very misleading in their resemblance to abdominal tumors.

Occasionally attacks of diarrhoea may occur in persons subject to habitual constipation. Such attacks may result from temporary catarrhal irritation of the bowels caused by impaction or excessive retention of the intestinal contents from food irritation, or other forms of irritation (purgatives), which act excessively on account of the retention of faecal matter and its hardened character. In cases of faecal impaction in the rectum, the real conditions may be obscured by a continuous diarrhoea caused by irritation of the mucous membrane by the retained mass.

The general condition of the patient suffering with habitual constipation may not be seriously impaired. In some cases, however, emaciation, loss of strength, and some anaemia may result from the gastric disorders that usually attend constipation, from the constant use of purgative medicines, and possibly from obscure toxæmic conditions.

Complications.—Among the intestinal complications are dilatation and pouching of the rectum and sigmoid flexure, formation of diverticula, volvulus, and inflammatory lesions in various parts of the bowel (rectum, colon, appendix). All of these will be separately discussed. Constant straining in constipation may lead to weakening of the abdominal wall and to the formation of hernia. The same cause, together with the pressure of masses accumulating in the rectum and the direct irritation occasioned by hard scybala, frequently leads to the development of hemorrhoids. Severe straining sometimes occasions thoracic diseases, such as emphysema of the lungs and aneurism. The onset of the latter is not infrequently traceable to such straining from constipation. Apoplexy may sometimes result from the elevation of blood pressure caused by severe straining.

Diagnosis.—The recognition of constipation usually offers no difficulty whatever, as the infrequency of the evacuations and their character are distinctive. Sometimes, however, there may be daily evacuations of comparatively normal amount, and apparently normal in other respects, in which a physiological test made with the use of charcoal or carmine will show that the time that elapses between the ingestion of a certain meal and the discharge of the residue is considerably prolonged. In other words, there is, in these cases, a latent form of constipation, due to the sluggish movement of the current; and there may be symptoms of more or less decided character. The recognition of such cases might prove difficult, if one did not recognize the possibility of such an occurrence. Fulness of the abdomen, general

sluggishness, a tendency to headaches, digestive disturbances, oppression, and other similar indefinite symptoms may suggest the existence of such latent constipation; and this may be suspected, also, in cases in which there is visceroptosis.

Another direction in which the diagnosis of constipation frequently offers difficulties is in the determination of the cause of the condition. The existence of distinct gastric symptoms antedating the tendency to constipation may suggest that digestive disturbance is mainly operative. A study of the patient's diet may bring out that a too monotonous diet or one lacking in fluid elements, or, on the other hand, foodstuffs that by irritation provoke intestinal spasm lie at the basis of the condition. The condition of the abdominal walls, a tendency to gaseous distention, and other indications of sluggish peristalsis may show that constipation has resulted from intestinal atony. Hemorrhoids or fissure, pelvic diseases, displacements of the uterus, or the presence of abdominal tumors may indicate that the disorder has resulted from reflex atony or spasm of the bowel; or, in other cases, from direct pressure upon it. Finally, general nervous conditions, such as hypochondriasis, neurasthenia, or other depressive conditions, may suggest the fundamental importance of the central nervous system in the etiology. In these instances, careful consideration of the possibility that the nervous conditions are secondary to the constipation must be remembered in attempting to determine the sequence of events.

No satisfactory treatment can be instituted until the proper causation has been investigated and determined.

Treatment.—The most important element in the treatment of this common condition is the establishment of the habit of regularity in the evacuation of the bowels at a certain time of the day. In discussing the causes of the condition, it was shown that the normal regularity of evacuation is due to the fact that the residue of food and the admixture of bacteria and secretions which constitute the fæces reach the lower bowel at a certain interval after the ingestion of food, and there stimulate the mucous membrane and bring about a desire for evacuation. A habit of resisting this desire may gradually cause a blunted sense, which eventually occasions more or less obstinate constipation. A process of reëducation is frequently effective, and the most natural and desirable method of treatment in the condition is the establishment of a habit of regularity. For some time no result may be achieved, but by repeated efforts at evacuation at a certain hour—preferably that which seems to be the normal time (that is, the morning)—will gradually prove effective.

In some cases it seems desirable to choose the evening rather than the morning, especially when local rectal conditions, such as hemorrhoids, are present. In these circumstances the retention of fæcal matter in the lower bowel during the night may occasion an irritation that may cause a reflex spastic constipation.

Simple measures may be used to supplement the effort at reëstablishing the normal regularity. Among these, the use of small enemata of plain water or soapsuds, with or without the addition of a little glycerin, and glycerin, gluten, or soap suppositories, may be recommended. All such aids, however, must be used with caution, as the bowel readily accustoms itself to artificial assistance. Sometimes vigorous kneading of the abdomen, carried out by the patient himself, may aid in the desired result.

Exercise.—The condition of the abdominal muscles was referred to as being important in maintaining regularity. The constipating effect of enforced rest in bed, or of an enforced sedentary life in those who have before been regular, indicates the importance of this factor. Exercise of the abdominal muscles in particular is important, although the muscular system as a whole must be considered, since its normal healthful condition has a bearing upon the function of the bowels, as on other visceral functions.

The form of exercise must vary according to the physique, character, and occupation of the patient. In many, the institution of gymnastic movements and the use of pulley weights, rowing machines, or other apparatus that will enable the patient to obtain vigorous exercise, particularly of the muscles of the abdomen and trunk, answer the purpose. In others, a better result is accomplished by directing the patient to occupy himself with such labors as will secure the proper amount of exercise. In obstinate cases, passive exercise or massage must be employed. The patient may knead the abdominal muscles in the direction of the large intestine, or may use a cannon ball covered with felt or leather, which is rolled about the abdomen in the same direction as that used in massage. The weight of the ball may vary from two or three to ten pounds. Various implements have been devised for producing vibrassage, and these may give useful results in the treatment of constipation. Active stimulation over the head of the colon and sigmoid, and a continuous treatment along the line of the large intestine, will give the best results.

A skilled manipulator combines all these forms of exercise by using stroking, kneading, and rotary movements and vibrations on the points indicated or along the entire length of the colon. Sometimes it is well to combine with the manipulations of a masseur high injections of plain water, which still further stimulate intestinal contraction.

Electricity and hydrotherapy have been used to supplement exercise or other forms of direct treatment. A slowly or rapidly interrupted faradic current may be applied over the colon, beginning at the cæcum, and following the entire length of the large intestine. Galvanism has also been employed, the current being interrupted from time to time. Ordinarily, in the use of either faradic or galvanic electricity, one electrode is placed under the back, while the other is applied to the surface of the abdomen. Occasionally advantage may be obtained from the use of an electrode inserted into the bowel. This method of treatment, however, is much less desirable, as it is more difficult and unpleasant.

Baths of various sorts have been employed to stimulate the abdominal muscles, as well as the intestinal peristalsis. Cold douches, applications of wet compresses to the abdomen, and general hydrotherapy may be useful in promoting improved conditions of the abdominal muscles.

Diet.—It is impossible to make any general statement regarding the character of diet that should be prescribed for patients suffering with this condition. In general, it may be said that such forms of food as furnish a considerable amount of residue are likely to prove useful, while those foods which are susceptible of complete digestion and absorption tend to occasion constipation if they form too large a proportion of the whole dietary. It has, therefore, become customary to prescribe for patients with constipation various vegetable foods that leave a large residue. Other things being equal, this plan proves satisfactory; but sometimes unabsorbed portions of

food may act as irritants and tend to increase, rather than to counteract, the condition. In the same way, fruits, berries, and the like, which contain seeds or other non-absorbent constituents, may be helpful or hurtful, according to the nature of the case. All kinds of fruits have been advised, and frequently aid in combating the trouble. Sometimes, however, by causing disturbance of gastric digestion, mainly through their acidity, fruits may have an opposite effect. Sweets, honey, milk sugar, and other saccharine or carbohydrate foods sometimes prove efficacious. Stewed fruit at night, or a few dried plums, may have a useful effect. Milk sugar has been largely employed in the same way.

In some cases the regular administration of water in increased amounts, and especially one or two glasses of warm or cold water in the morning before breakfast, may be helpful.

Certain inert substances, such as agar-agar, which swells from imbibing water and is not digested or absorbed, paraffin and liquid vaseline are sometimes added to the diet with advantage. The regular use of olive oil in doses of from a teaspoonful to an ounce after each meal acts somewhat similarly. Various kinds of bread made of coarse flour or containing bran may aid by supplying a certain degree of desirable irritation.

As a general rule, however, it is desirable to regulate diet in such a way that it shall preserve a normal admixture of different sorts of foodstuffs, rather than an excess of any one sort, no matter how much this may, in the individual case, facilitate intestinal activity. Certain foods have a recognized constipating effect. Among other things, acid wines or tea, on account of the astringent effect of the constituent tannic acid, are undesirable. Milk is usually constipating, but only when it forms a considerable portion of the whole diet, and when, as a rule, the amount of residue left after digestion is inconsiderable and insufficient.

Medical Treatment.—Medicinal agents should be used in the treatment of constipation only when a regulation of the daily life of the patient, or of his diet, and the employment of various physical means of stimulating peristalsis have failed of the desired result. Undoubtedly, the common cause of increasing constipation is the employment of remedies without reference to the individual conditions, and usually without medical advice. Often, however, some kind of direct medical treatment seems necessary; and drugs may be required. These may be divided into those which exercise a helpful influence on digestion and peristalsis, without having a direct laxative effect, and those which are laxatives in a narrower sense.

Among the remedies that are correctives of digestion and stimulate peristalsis without irritating, *nux vomica*, *belladonna*, *physostigma*, and digestive ferments may be considered. In cases of constipation associated with gastric sluggishness or chronic gastritis, such a combination as the following may prove useful:

R—Extract. nucis vomicæ (exsic.)	gr. vj
Sodii bicarb.	gr. c
Extract. pancreatic.	ʒj
Ft. in caps. No. xxiv.	
Sig.—One before each meal.	

Such a combination stimulates gastric activity and, by supplying a starch digestive ferment with an alkali, aids a more satisfactory starchy digestion before the acidity of the stomach has ended this process. The addition of

a little extract of belladonna or physostigma to the formula given may aid intestinal peristalsis.

In some cases a full dose of pepsin after meals seems to act as a laxative. Certain non-astringent stomachics, such as cinchona derivatives or strophanthus, may be useful in stimulating peristalsis. In some cases a small dose of tincture of belladonna, such as one to three drops of the tincture at night, may suffice to aid the condition.

Of the direct laxatives, aloes and cascara are probably most generally useful. The well-known combination of aloes with belladonna and strychnine in pill form, to be taken at night, is useful, and if carefully controlled by other measures rarely requires such increases in the quantity of the remedies as to make it distinctly undesirable. Aloes may also be combined with podophyllin, extract of rhubarb, or cascara.

Cascara, in the form of the fluid extract or solid extract, or of various elixirs, is a favorite remedy, and, like aloes, does not seem to increase the tendency to constipation. It is difficult to determine without experiment the required dose, individuals differing very widely in this respect. The combination with aloes or other purgatives often acts more happily than the drug without addition.

Senna is a remedy widely used in domestic practice, and forms the basis of many proprietaries. An old practice used in many institutions is to administer stewed prunes, to which a few leaves of senna have been added; and senna teas, decoctions, etc., are extensively used. The official confection of senna, or one containing senna, rhubarb, sulphur, and glycerin, taken before bedtime, may be used. The following formula is an old one:

R̄—Sennæ	℥ss
Extr. glycyrrhizæ,	
Sulphuris	āā ʒij
Pulv. rhei	ʒj
Glycerini	q. s.
Ft. confectio.	
Sig.—A small portion at night.	

Sulphur itself may be used as a laxative, although its potency is rather limited.

Various remedies may be used from time to time in emergencies, as when an acute exacerbation requires more vigorous measures. In such circumstances, jalap, in the form of compound jalap powder, compound licorice powder, calomel, or castor oil, may be employed.

Saline waters of various sorts, such as those of Marienbad, Carlsbad, Bedford, or Saratoga, may be useful. One or two drams of saturated solution of magnesium sulphate or Rochelle salt have a similar effect. As a rule, these waters are most efficacious when taken before breakfast or at night. The continued use of waters in regulated amounts may have a permanently useful effect, correcting digestion or catarrhal conditions of the gastro-intestinal tract, but the unrestricted use of any kind of saline water must be condemned.

Rectal medication is often highly beneficial, and in cases in which some form of remedial agent seems to be necessary, the substitution of enemata, suppositories, etc., for remedies given by the mouth has the effect of preventing the development of a habit. In France and some other countries the habit of employing enemata regularly or daily is quite common. It is not,

however, harmless, as the effect upon the mucous membrane of the lower bowel sometimes proves distinctly deleterious. As temporary expedients, however, enemata of plain water, saline solution, soapsuds, or more powerful combinations, such as solutions of sulphate of soda, Rochelle salt, or other salines, may be employed. In some cases, the injection of a small quantity of olive oil at night, to be retained until morning, has a useful effect, particularly in cases in which larger enemata are irritating. In cases of spastic constipation, such injections of oil or enemata of large quantities of oil may be employed. The addition of glycerin to enemata render them distinctly more powerful, and especially efficacious in acute cases.

Sedatives may be useful in the treatment of constipation when the condition is more or less dependent upon spastic states of the bowel. In these circumstances the milder sedatives, such as bromides, valerian, and especially asafoetida, given in the form of pills, each containing gr. ij, or administered by enemata in the form of milk of asafoetida, serve a useful purpose. It is well known that in the obstinate spastic constipation of lead poisoning opium sometimes has a paradoxical effect, causing relaxation of the spasm and relief of the constipation.

Some attempts at correcting constipation due to a deficiency of microbic action have been made by the employment of cultures of microorganisms, such as the *Bacillus coli*, lactic acid bacilli, etc. These methods, however, have not proven especially valuable.

Surgical Treatment.—Some attempts have been made to correct constipation by eliminating the large bowel from the intestinal tract. Among others, Lane has practised anastomosis of the small intestine with the sigmoid flexure or the rectum. Complete removal of the large intestine has been practised, but is obviously too radical a procedure to warrant recommendation. Even the more conservative forms of surgical treatment are justifiable only in cases of unusual obstinacy.

IDIOPATHIC DILATATION OF THE COLON.

This term is used to define certain cases of more or less extensive and persistent dilatation of the colon, probably dependent upon structural abnormalities, and not due to ordinary obstructions and to consequent accumulations of gaseous or faecal contents.

The cæcum, colon, and sigmoid may be enormously distended throughout their entire lengths. In some cases, however, the sigmoid alone is involved, and may be so greatly enlarged that it fills the entire abdominal cavity, pressing other viscera out of the way. The diameter of the bowel may reach as much as eight to ten inches. When the colon is involved, the abdomen is filled with the enormously distended ascending and descending colon, occupying either half of the abdominal cavity, which itself is so greatly enlarged that the patient presents the appearance of a monstrosity. Formad's celebrated case had been exhibited in various places under the name of the "balloon man."

Etiology.—A distinction may perhaps be made between cases that occur in young children and those that are met with in the elderly or aged. The former group probably represents cases in which some congenital defect, such as great inertness of the bowel, has been the occasion of the dilatation,

which gradually progresses until the patient succumbs. In the cases occurring after childhood, contributing causes, such as partial obstruction, habitual constipation, and weakening of the bowel by chronic inflammatory affections, probably more frequently play a part. In a case reported by Treves the dilatation was due to congenital narrowing of the rectum; and this author concluded that idiopathic dilatation is usually due to obstruction with resulting faecal impaction. This view has not been sustained by later investigations, such as those of Crozer Griffith, who analyzed the postmortem findings in eighteen fatal cases in children. Marfan has suggested that a congenital abnormality in the form of the sigmoid flexure might be the cause, and a number of cases have been described in which the sigmoid was abnormally long and therefore distorted. Other causes of impediment in either the sigmoid or the rectum may occasionally play some part in the causation. Some form of muscular weakness of the entire bowel has also been assumed as the basis for the development of this condition; but idiopathic dilatation is not the result merely of atony of the bowel, as the walls are invariably found hypertrophied.

The important features in the anatomy of idiopathic dilatation are the great distention, a thickening of all the coats of the bowel, and especially an hypertrophy of the muscle. The mucosa is usually swollen, congested, and the seat of numerous small or large ulcerations. As a rule, it contains large quantities of faecal matter; in Formad's case the contents of the colon weighed forty-seven pounds.

Symptoms.—The most conspicuous feature of the condition is the abdominal enlargement, which, on examination, proves to be due largely to accumulated gaseous contents. Griffith and Fitz point out that the chronic phantom tumor of older writers was probably the condition now under consideration. The abdominal distention may be uniform and globular, or may be more decided on one side—particularly on the left side, when the sigmoid is especially involved. The coils of intestines may be visible through the abdominal wall, and peristaltic waves may be seen passing along them.

Interference with breathing and with the circulation is commonly the result of the upward displacement of the diaphragm. The patient complains of abdominal distress, but acute pain is usually wanting. Gastric disturbances, headaches, and intense constipation are usually encountered. Sometimes there are paroxysms of colicky pain, and occasionally attacks of diarrhoea may set in, especially toward the end of life. In some instances the bowels have been regular throughout, although there was nevertheless a gradual accumulation of retained faecal matter. The patient suffers with increasing malnutrition, which finally may become extreme; and death may be due to inanition and exhaustion. In other cases a fatal termination is due to volvulus of the sigmoid or to perforation of the bowel at a point of ulceration. Sometimes the fatal result appears to be due to the extensive associated colitis.

The clinical course of the condition in congenital cases begins within the first few days of life. The abdomen begins at once to enlarge, and increases progressively. At the same time, obstinate constipation attracts attention, practically no faecal matter being discharged, except at long intervals. In some cases a history has been obtained that the child had had but few bowel movements since birth, although it was then some months or a year

or more old. The child usually dies early, as the result of the conditions described. In Griffith's series only three out of twenty-four cases reached adult years. In other cases the symptoms do not begin until a year or more after birth. Milder forms, and sometimes even extreme cases, like that of Formad, may go on to adult or middle life.

Treatment.—In some cases the administration of purgatives may cause evacuation of retained contents; but this is followed by a speedy recurrence of the condition, and is attended with little relief, even for a time. The same unsatisfactory result attends the use of enemata, colonic douches, etc. In a number of cases surgical treatment has been employed, and in a few has been followed by satisfactory results. The operation most generally employed has been colostomy, which in several cases has been quite successful. More extensive operative procedures, such as excision of parts of the bowel, have been resorted to with variable results. Richardson's case, in which excision of the sigmoid flexure was followed by the formation of a new sigmoid, is instructive in illustrating that inherent structural defects may lie at the basis of the etiology in some instances. Probably in most cases operation has been undertaken at too late a day. Fitz insists upon radical operations because of the insufficiency of mere colostomy, and more especially because of the deplorable condition of a patient who has an artificial anus.

DIARRHŒA.

Definition.—The term diarrhœa is usually applied to the too frequent discharge of more or less fluid stools. In many persons two or three daily evacuations of the bowels occur without disturbance of health, and without any marked alteration in the character of the fæces from the normal. This does not constitute diarrhœa in the ordinary acceptance of the term. Usually, however, when more than one daily movement occurs, the fæces are also less solidified; and although active symptoms may be wanting, the condition is really one of moderate diarrhœa. It is important to recall, also, that in some cases repeated evacuations of small quantities of fluid or semifluid fæcal matter may occur in conjunction with an actual obstructive condition of the bowels and fæcal impaction.

Etiology.—As the essential condition in diarrhœa is the presence of an abnormal amount of water in the fæces, the causes will be found to be such as occasion the rapid evacuation of the intestinal contents, with consequent reduction in absorption or an oversupply of water or increased secretion from the mucous membrane of the bowel.

Normally, the contents of the upper bowel are fluid, and pass from the stomach to the cæcum in from two to six hours. The contents of the large intestine become progressively more inspissated, as the result of the absorption of water, the duration of their passage through this part of the intestinal tract being from twenty to twenty-four hours. Any conditions that hasten the movement of the contents, especially in the large intestine, therefore, tend to cause the fæces to be more liquid. The same result may occur from a variety of conditions that disturb the process of absorption and thus prevent the normal inspissation of the bowel contents. Sometimes conditions that influence the absorptive power of the mucosa, such as catarrhal irritations, are due to causes that, at the same time, occasion an excretion of

considerable quantities of fluid; and the same agents may also stimulate active peristalsis, and thus hurry the contents of the bowels through the tract.

The forms of diarrhœa may be classified as those without definite organic lesion and those associated with various intestinal diseases. In the latter group the condition is to be looked upon as symptomatic, and will be more appropriately considered in connection with the various diseases that cause it.

Diarrhœa Due to the Excessive Ingestion of Water and Other Fluids.—Sometimes the mere drinking of excessive quantities of water may be the cause of looseness of the bowels. In some individuals in whom there is normally a constipated condition excessive water drinking may correct the difficulty or even produce a temporary diarrhœa. The drinking of other fluids, such as wines, malt liquors, etc., may act in the same way, although there is probably here an associated cause, the digestive disturbance resulting from the overuse of the fluids named. Diarrhœa is sometimes produced by the use of large quantities of oil, butter, or fatty food. In this case the amount of unabsorbed fat may itself occasion looseness, or digestive disturbance and fermentative conditions in the intestine may result from the excess of fat consumed, and the fatty acids or other products of decomposition may act as irritants.

Diarrhœa Due to Food.—Various foods may occasion diarrhœa, through their mechanically irritating character, through their ready decomposition in the gastro-intestinal tract, and sometimes through their mere bulk. It is well known that fresh fruits, certain vegetables containing abundant cellulose, and berries containing small seeds may, in some individuals, readily provoke an attack of diarrhœa that can be explained by the mechanical irritation occasioned by the unabsorbable residue. Other foods, such as fish, shell-fish, cheese, milk etc., may easily undergo decomposition in the bowels or may contain preformed products of decomposition, particularly in hot weather, and thus may excite more or less acute or violent diarrhœa. Various microorganisms have been found in association with cases of this sort. These are properly considered in connection with the subject of food-poisoning.

Not rarely mere excess of food may set up a diarrhœa as a result of derangement of the gastric and upper intestinal digestion and secondary bacterial fermentations.

Diarrhœas Due to Purgative Substances.—Any of the various purgatives used in practical medicine may occasion active diarrhœa. The majority of these act upon the muscle of the bowel and its nervous mechanism without influencing the secretions or absorptive processes. Their effect is simply to stimulate peristalsis, especially that of the large intestine, and thus to hasten the contents of the bowels through the tract before proper absorption can take place. The salines, however, increase watery secretion from the intestinal mucous membrane; and some of them, in addition, exercise a stimulating effect upon peristalsis. The diarrhœas resulting from irritation by certain foods, particularly those in which bacterial decomposition has occurred, are often similar in character to those caused by neutral salts, being very copious and watery, from the excessive secretion of fluid from the intestinal mucosa.

Diarrhœa Due to Nervous Influences.—A variety of conditions may excite increased peristalsis by directly affecting the nervous mechanism of the

bowels. The influence of shocks, fright, fear, and various kinds of excitement is well known. In some diseases attended with disorders of the nervous system, like Graves' disease, hysteria, or neurasthenia, attacks of diarrhœa or continued looseness of the bowels may be met with. In some persons the nervous mechanism of the intestines appears particularly susceptible, and slight influences, such as the emotional conditions already named, exposure to cold, or various forms of irritation ordinarily having little effect, may readily increase peristalsis. The condition known as morning diarrhœa, in which several evacuations occur in the early morning, after which the bowels remain quiet until the next day, may be explained by assuming the existence of an irritable state of the nervous mechanism in the lower bowel, and the consequent prompt evacuation of contents that usually reach this portion of the tract twenty-four hours after the beginning of ingestion. After the evacuation of the lower bowel conditions become quiescent until the remains of the food of the succeeding day have reached it.

Diarrhœa Due to Diseases of Other Organs and Disordered Conditions of the Blood.—A tendency to diarrhœa may occur in some diseases of the stomach, heart, or kidneys, and as the result of other affections not involving the intestinal tract. In diseases of the stomach, when excessively acid chyme is discharged into the upper bowel, the resulting irritation may provoke active peristalsis; and the insufficiently neutralized chyme may be hurried through the intestinal tract, provoking increased secretion from the mucous membrane and excessive peristalsis throughout the whole length of the bowel. In some cases of heart disease, congested conditions of the mucous membrane of the bowel may occasion a proneness to diarrhœa, or may cause it directly by increasing intestinal secretion. The association of diarrhœa with renal disease is more important than either of the others. In this case the vicarious excretion of urea from the intestinal mucous membrane and the decomposition of this, with the production of ammonium, occasion irritation, sometimes of intense character. In all cases of severe kidney disease the development of diarrhœa must be regarded with suspicion. Attention has been frequently called to the association of diarrhœa with malaria. This has been attributed by some to conditions of the blood in that disease, while others, as Brunton, believe it to be a nervous manifestation.

More or less intense diarrhœa may be associated with various infections not necessarily involving the intestinal tract itself. Thus, in cases of septi-copyemia and pneumonia, and in various other infections with lesions at a distance from the bowel, diarrhœa may occur. There is reason to believe that such attacks are due to the elimination of toxic matters from the intestinal mucous membrane, just as urea is eliminated in cases of disease of the kidneys. Recent experiments have shown that the toxins of such organisms as the *Bacillus dysenteriae* injected hypodermically may be eliminated through the bowel, causing secondary lesions of the mucous membrane. The same is true of injections of cholera toxin. Even in infections of the intestinal tract, therefore, such as cholera, dysentery, etc., the attending diarrhœa may be due to the toxic state of the blood and the secondary elimination of the toxins through the mucous membrane of the bowel.

Diarrhœa Due to Organic Diseases of the Bowel.—In connection with enterocolitis and other catarrhal and inflammatory conditions of the mucous membrane, in the descriptions of the various ulcerations of the bowels and of

malignant disease, diarrhœa will be discussed as a symptom of importance. In the present section, however, only functional disturbances and such as are attended with but a minimal degree of organic disease will be considered.

Bacteriology of Diarrhœa.—Bacteria and their products are sometimes the direct causes of diarrhœal conditions. A considerable variety of micro-organisms may be concerned in the production of diarrhœa, and it is difficult to determine with certainty in many cases which of the abundant intestinal flora is mainly operative. Certain facts, however, have been learned by observations of the predominant organisms in different cases, by the discovery of forms in abundance that normally are not present and that have been traceable to tainted food, and by the application of agglutination tests to the serum of individuals suffering with diarrhœa.

Certain of the normal organisms of the intestines to which the system has grown accustomed may under peculiar circumstances undergo unusual multiplication or increase of virulence, so that they become pathogenic. Among these the *Bacillus coli communis* is most important. The fact that this organism is largely present in cases of diarrhœa does not necessarily imply its etiological relation, although other considerations may establish such a connection. There is little doubt that under some conditions diarrhœal diseases are caused by this group of organisms. Preceding irritation of the bowels due to other causes, digestive disturbances, and errors in diet, may be the occasion of active multiplication of the normal colon bacillus, and may probably also increase its virulence.

Closely allied organisms, such as the *Bacillus enteritidis* of Gaertner, and members of the intermediate group, the paratyphoid and paracolon bacilli, are important causes of certain diarrhœal conditions. Diarrhœa due to the *Bacillus enteritidis* has been found in some cases of meat poisoning occurring endemically or in epidemics, and sometimes also in cases not directly traceable to the consumption of tainted meat. The *Bacillus dysenteriae* of Shiga and the varieties described by Flexner, Park, His, Harris and others are the specific cause of certain types of dysentery and of diarrhœa. In some cases, especially in the diarrhœas of childhood, streptococci and staphylococci appear to be the active agents, and may cause intense forms of diarrhœa or enteritis. Among less frequent causes are various others of the organisms named as inhabiting the intestinal tract normally (see Bacteriology of the Intestinal Tract), which may operate by their direct action or by causing fermentative processes with the liberation of fatty acids and other irritant substances that secondarily occasion intestinal disturbance and diarrhœa. The *Bacillus pyocyaneus*, Friedlander's bacillus, the *Bacillus botulinus*, and other forms occur more rarely. Certain organisms of specific diseases, such as typhoid fever, tuberculosis, syphilis, etc., occasion characteristic lesions and often an associated diarrhœa. These forms are not properly under consideration in this place.

Diarrhœa is sometimes occasioned by protozoa, including amœbæ and a variety of flagellate and ciliated forms.

Symptoms.—The clinical manifestations of diarrhœa depend largely upon the nature of the condition and the part of the bowel involved.

In cases of irritation of the upper intestine due to overloading with food or functional derangement, diarrhœa may not occur if the peristalsis of the colon remains normal; but usually an increase of activity occurs in this part of the tract as well, and some looseness results. In such cases the stools

are likely to present features that indicate that the upper bowel is at fault. Undigested food, with the admixture of considerable mucus, rendering the fæces pasty, sticky, or viscid, and a yellowish or greenish coloration, due, in part, to unaltered bile and largely, often, to pigment derived from bacterial action, are characteristic. Excessive acidity from insufficient neutralization in the upper bowel, and from secondary fermentative processes, may render the discharges highly irritating, and may occasion secondary inflammatory conditions of the rectum, anus, or even of the skin externally. This is commonly met with in diarrhœas of this type in childhood.

In cases of widespread irritation of the bowels, involving both the large and the small intestine, more copious watery discharges are the rule. There is usually an admixture of fæcal matter, and the color of the movements may vary from a dark brown to a light yellow; or, when abundant serous outpourings occur, as in choleriform types, almost watery discharges are seen.

When the large intestine is particularly involved there is usually considerable mucus of whitish color, and more frequently in masses than in cases involving only the upper intestine. It may be seen as jelly-like particles or as large accumulations; and sometimes, alternating with the discharges of ordinary diarrhœal type, the evacuation of almost pure mucus or mucus streaked with blood may be encountered. The special form of disease known as mucous colitis will be discussed in a separate section.

The subjective symptoms in cases of moderate diarrhœa may be very slight. As a rule, however, when the small intestines are involved, colicky pains and abdominal tenderness are usual. In some instances this may be intense, and the patient may become prostrated from the severity of the pain, as well as from the loss of water and other causes. In cases of diarrhœa involving special irritation of the lower bowel, local tenderness in the left flank or above the pubis may signify involvement of the descending colon and sigmoid. In these cases, also, tenesmus is not unusual.

Soon after the onset of diarrhœa a certain degree of weakness or prostration is usual. In mild cases this is of little consequence. In severe forms extreme prostration or complete collapse may occur. Pallor, coldness of the extremities, sweating, and sometimes all the indications of collapse, may occur. The circulation becomes enfeebled; a little cyanosis may be present; and in children, sinking of the eyes and fontanelles, pinched features, and other indications of the Hippocratic facies are seen.

Fever is rarely found in mild cases, but slight elevations are not unusual in the more severe. On the other hand, the prostration may be such that the temperature soon becomes subnormal.

Disturbance of the stomach sometimes attends diarrhœa, either as a result of the original dietary disturbance or other irritation that has occasioned the diarrhœa, or as a reflex result of pain and abdominal irritation.

The clinical course of cases of diarrhœa varies with their cause and nature. In ordinary instances due to digestive disturbance a few evacuations succeeding each other at intervals of a half hour to several hours may terminate the condition. In more severe cases, and particularly when more intense irritation of the intestines has occurred, the diarrhœa may increase in severity for a day or two, or may reach its maximum only after several days. In such instances the stools tend to become more and more watery as the contents of the bowel are evacuated, while in cases in which the large bowel

is involved there may be, in the later stages, only discharges of mucus. In most instances the entire duration is not above several days, unless secondary enteritis or colitis has complicated the condition.

Diagnosis.—The diagnosis of this condition is mainly concerned with the recognition of the cause. Ordinary diarrhœa must be distinguished from various forms of specific intestinal disease or general infections accompanied with intestinal manifestations. Frequently it is possible to connect the attack with some dietary indiscretion, and thus to establish a diagnosis.

Treatment.—Prophylaxis is of great importance. The avoidance of irritating or tainted foods, particularly during hot seasons of the year, when decomposition is apt to occur, is the most important measure to be adopted. Uncooked fruit, shell-fish of various sorts, berries, and cold drinks are among the articles of diet to be scrutinized with greatest care. Residents of hot climates have found it advantageous to wear an abdominal band of flannel or some woven material, to secure warmth and protection, particularly when cool nights succeed hot days.

Local applications of heat over the abdomen, in the form of hot-water bags, hot fomentations, and binders of various sorts, are often quieting in the onset of attacks of diarrhœa, and may moderate the severity of the attack by controlling peristalsis.

Diet.—The diet during an attack of diarrhœa should be extremely moderate in the beginning, and often it is necessary to avoid food altogether. Later, and particularly in cases in which prompt control is not secured, a cautious increase of food is requisite, lest the patient's strength be too greatly impaired. Thirst, which may be complained of severely, should be controlled by the swallowing or eating of small bits of ice and the sipping of small amounts of cold water. Frequently, merely holding water in the mouth is satisfying. Albumin water, barley water, rice water, or toast water may be used to quench thirst and, at the same time, to supply a certain amount of nourishment. If the stomach is irritable, these liquids may be better tolerated than plain water. After preliminary evacuation of the bowels has been secured, a diet of pasteurized or boiled milk, given in small quantities, or milk with well-cooked farinaceous foods, such as arrow-root, rice boiled in milk, milk toast, etc., may be given. Later, animal foods, including soft-boiled eggs; gelatin preparations, such as meat jellies; and, still later, scraped beef or minced chicken, may be allowed. It is usually advisable to avoid giving vegetables until the signs of gastro-intestinal irritation have practically subsided.

Medicinal Treatment.—After the onset of diarrhœa, the first consideration in the treatment is the evacuation of irritating material, if the spontaneous discharges are not enough to carry these off. Nearly always it is desirable to use some unirritating and rapidly acting purge, such as castor oil. It is a common practice to add to this a small amount of tincture of opium or paregoric, to allay irritation and prevent unnecessary griping. Other purgatives, such as calomel, salines, magnesia, and the like, may be used instead of castor oil; but they are less prompt in their effects. After evacuation of the irritants the use of sedatives is desirable. Bismuth powders, to which may be added pepsin, if digestive disturbances are present; chalk mixture, cerium oxalate, lime-water, or other alkalies may be used to quiet intestinal irritation and neutralize acidity. A combination of chalk mixture

with paregoric and some astringent, like tincture of kino or catechu, is frequently used.

When cramp-like or colicky pains are present, some form of sedative and carminative may be added or substituted for the remedies named. Chlorodyne or improvised mixtures containing other carminatives, such as spirit of chloroform, compound tincture of cardamom, or spirit of ginger, with opium, may be employed for this purpose.

When intense pains occur and collapse threatens, the timely use of a hypodermic injection of morphine, followed by the various remedies already named, may cut short an attack that promises to be serious. Sometimes suppositories of opium may be substituted for these, or repeated doses of paregoric or of tincture of opium may be given by the mouth.

Sudden diarrhœas, attended with marked evidences of intestinal irritation, should be treated with promptness; and active remedies, astringent, sedative, and protective, should be given early. As a rule, the evacuations are so free that little is gained from a preliminary administration of purgatives; and these may, at times, be actually harmful. Bismuth in fairly large doses (gr. xxx), in the form of the subnitrate, subgallate, or salicylate, with vegetable astringents, such as kino, catechu, or hæmatoxylon, repeated doses of opium, and an occasional hypodermic of morphine, may be given. Mineral astringents sometimes have a greater controlling effect than the vegetable forms. Small doses of subacetate of lead, nitrate of silver, or sulphate of copper may be employed. The so-called intestinal antiseptics, like salol, betanaphthol, guaiacol carbonate, etc., have little effect in such acute cases, although they may be useful in later stages or in less active attacks.

When diarrhœa becomes obstinate or chronic, persistence in the use of the same kind of treatment, using small doses of the various drugs named, will gradually control the disorder. Diet here, however, plays a more important role than does the medicinal treatment of the disease. No general rules can be laid down regarding the most desirable diet, as individual peculiarities play an important part, and therefore require individual consideration. In most cases the use of pasteurized or boiled milk, with or without small amounts of farinaceous food, proves most suitable. In some persons, however, milk is not tolerated; and even forms of food prepared with milk may protract the diarrhœa. In such individuals, eggs and rare meat, with a farinaceous diet, may prove more acceptable. Occasionally the use of some astringent water, like the Rockbridge alum water, aids in controlling a persistent diarrhœa.

Nervous diarrhœas are best treated with remedies directed to the general nervous system and careful regulation of the diet, so that any undue taxing of the digestive functions may be obviated.

Diarrhœas attended with irritation of the lower bowel sometimes require local treatment. In the acute stages, if tenesmus, pain, and other local symptoms are marked, relief may be obtained from ice suppositories or from the injection of small quantities of cold water or larger amounts of starch water containing tincture of opium. When a residual catarrhal condition persists, the cautious use of colonic injections of weak solutions of nitrate of silver (1 to 5000) may be useful; but repeated injections and stronger solutions may easily do harm.

ACUTE ENTERITIS.

Definition.—This term may be employed to designate acute catarrhal inflammation of the mucous membrane of the small intestine, as well as the upper portion of the large bowel. The terms acute inflammatory diarrhœa, enterocolitis, and ileocolitis are synonymous with that here preferred. It is impossible to distinguish this condition sharply from irritative diarrhœas due to digestive disturbances and certain infectious diseases with intestinal manifestations. In the former group are cases in which actual enteritis results from the highly irritating character of products of intestinal fermentation. In the latter group are many infections often situated at a distance from the intestinal tract, in which complicating enteritis results from irritating toxins in the blood, excreted into the bowel. There must also be remembered the specific infectious diseases, such as typhoid fever, cholera, dysentery, etc., in which enteritis is an important lesion.

Etiology.—Individual susceptibility often plays a part in the causation, certain persons being exceedingly liable to this form of disease. Enteritis is more common in hot weather and in hot climates than at other times or elsewhere, owing to the greater likelihood of the contamination of food in these circumstances, and because certain easily tainted foods like fruit are more commonly used. As in childhood, chilling of the surface during cool nights following hot days plays some part in the etiology.

The direct cause of enteritis is usually some chemical irritant contained in food. The irritants, however, may reach the intestines through the circulation. They are quite varied, depending upon the character of the food, its preparation, and its preservation. Sometimes, although wholesome when swallowed, excessive quantities of food or digestive disturbances may occasion fermentation in the intestinal tract and the formation of irritants that directly excite a catarrhal inflammation. Sometimes articles of food, like meat, fish, etc., may contain preformed toxic bodies such as ptomains or other products of bacterial action, which may occasion violent inflammations of the bowel.

Bacteria probably rarely cause catarrhal inflammation directly. Usually their relation is that of agents causing fermentative changes in food or intestinal contents, and the products of such fermentation are the immediate causes of the resulting enteritis.

Enteritis occurs as a more or less essential complicating lesion in certain infectious diseases, such as typhoid fever or dysentery; and as a less frequent accompaniment of other infections, such as pneumonia, the exanthemata, septicopyæmia, etc. The occurrence of diarrhœa as a symptom of uræmia has been referred to in the discussion of diarrhœa. In some cases there is an active enteritis in this condition.

Among other causes of enteritis must be named mineral poisoning, or poisoning by toadstools or other noxious substances. Overdoses of lead, mercury, arsenic, copper, and other mineral substances may occasion intense forms of enteritis with violent symptoms.

Pathology.—The mucous membrane of the bowel may be involved with almost equal severity from the stomach to the upper portion of the large intestine, but usually certain areas are more seriously implicated than others. The mucosa is swollen and reddened. Often the crests of the

valvulæ conniventes alone present an inflammatory appearance. The surface is usually covered with mucus, which may be blood tinged, and sometimes hemorrhagic extravasations occur in the mucosa. The lymphatic follicles are swollen and stand out prominently as light areas against the inflamed surface. The Peyer's patches may be swollen, and occasionally show small rounded ulcerations.

In exceptionally violent forms of enteritis, pseudomembranous or diphtheritic inflammations of the mucous membrane of the small and large intestine may occur. Sometimes this is primary, in the sense that no preceding infective lesion can be discovered; but much more commonly it is secondary to infections originating elsewhere. Thus, in the course of pneumonia, septicopyæmia, and other types of infection, secondary involvement of the bowel may occur. The same form of enteric disease may be met with in cases of chronic nephritis or uræmia, following cirrhosis of the liver and other conditions producing portal obstruction, and in association with obstructions of the bowel caused by carcinoma or other lesions. Sometimes an intense enteritis with pseudomembrane formation occurs as the result of mineral poisoning, such as is caused by lead, mercury, or arsenic.

In all these cases the mucous membrane is covered with a more or less extensive, dirty grayish or yellow deposit, appearing as a thin coating or as a firmly attached pseudomembrane, which simply coats the surface of the bowel or involves the mucosa completely. Areas of necrosis and exfoliation or deep ulceration may indicate the seat of the previous formation of a membrane that has been dislodged. Involvement of the lymphatic follicles may occur, and follicular or larger ulcerations may be met with. Surrounding the ulcerated areas the mucosa may be oedematous and swollen and injected, or the seat of hemorrhagic infiltrations.

Microscopically, the mucosa and submucosa are found swollen and congested, and a considerable increase of the lymphoid tissue between the glands is observed, while diffuse round-cell infiltration may attend the severer cases. The crypts of Lieberkühn are enlarged and the epithelial cells swollen and cloudy. Numerous goblet cells may be present. Superficial erosions from desquamation of the epithelium, and even ulcerations of the mucous surface, occur in the cases of decided severity. In pseudomembranous or diphtheritic types extensive destruction of the epithelium and glands, with replacement by necrotic material, is characteristic. Considerable inflammatory infiltration of the submucosa may occur in such cases. A rare form of enteritis is the phlegmonous type in which purulent infiltration of the submucosa is met with.

Symptoms.—The clinical manifestations of enteritis vary with the intensity of the cause and the part of the intestinal tract principally affected. In milder cases the symptoms are those of ordinary diarrhœa due to dietetic errors and functional disturbances. In several cases the manifestations are sudden and violent.

The stools, as a rule, are of ordinary diarrhœic character, liquid, brownish, and more or less offensive, according to the degree of putrefactive change taking place. In the beginning there may be merely pultaceous movements which evacuate the lower bowel; later the movements become more watery, and frequently contain a certain amount of visible mucus. When the large intestine is especially affected, the amount of mucus may become

considerable; but in most cases the discharges remain thin, watery, and brownish. When enteritis is due to irritants of great intensity, very frequent and abundant watery movements result; and an excessive diarrhoea, approaching in severity that of an attack of true Asiatic cholera, may occur. Sometimes constipation instead of diarrhoea is met with in cases of enteritis, the explanation of this phenomenon being that inhibition instead of stimulation of peristalsis has resulted from the irritation in the bowel. Undoubtedly many cases of acute constipation following indiscretions in diet and irritation of the upper part of the small intestine are produced in this manner. When actual enteritis is present, and especially when it is extensive, diarrhoea practically always results. In some cases of intense and localized enteritis paresis of the affected segment of bowel may occasion intense constipation or even complete obstruction of the bowel. (See Obstructions due to Motor Paralysis of the Bowel.)

The patient usually suffers with colicky pains and more or less abdominal soreness from the beginning. As a rule, this is of moderate severity, but sometimes it becomes extreme. Distention of the abdomen from gaseous accumulations may occur, and loud rumbling or gurgling sounds (borborygmi) are frequently heard. In some cases, owing to great severity of inflammation in a certain restricted part of the bowel, intense pain and tenderness may be found in a localized area of the abdomen. When the lower part of the ileum is involved the local symptoms may suggest appendicitis. In these cases rigidity of the abdominal muscles in the area of pain and tenderness, the occurrence of fever, of vomiting, and of leukocytosis may still further confuse diagnosis. The development of diarrhoea is usually the determining symptom in the establishment of a proper diagnosis, but this may be wanting in enteritis, and in exceptional cases is met with in appendicitis.

In the beginning, irritation of the stomach is usually met with, and vomiting may be a disturbing symptom at the onset. Later, this subsides, as the intestinal symptoms become more pronounced. Some gastric disturbance, however, usually persists. The tongue becomes coated and dry, appetite is wanting, there is more or less nausea or distress after eating, and frequently vomiting is easily provoked throughout the course. When the upper part of the small bowel is especially involved, active gastric symptoms may be a conspicuous feature of the clinical manifestations. Continuous nausea and vomiting and localized pain in the upper part of the abdomen are marked. Sometimes moderate jaundice indicates a special involvement of the duodenum and interference with the outflow of bile.

Fever is more or less high from the beginning, some degree of temperature rarely being absent. The temperature, however, bears no relation to the severity of the intestinal manifestation, being sometimes high and persistent when intestinal symptoms are slight, and on other occasions sinking to the normal or to a subnormal level when diarrhoea is excessive. In extensive enteritis of moderate severity a continuous febrile temperature resembling that of typhoid fever in degree and character may be encountered, and the diagnosis is therefore at times quite uncertain.

The patient suffers with loss of strength and general debility, in proportion to the degree of irritation and the intensity of the diarrhoea. In cases in which large, watery evacuations are frequently repeated the patient may fall into a state of rapid prostration or collapse. In such instances the surface

becomes cold and moist, cyanosis of the extremities may be noted, or there may be the complete picture of collapse.

The physical signs, as a rule, indicate nothing beyond a slight abdominal distention, with more or less tenderness on pressure. In some cases, however, marked tympany is observed.

The secretions become scanty, the urine particularly being reduced in quantity, and often presenting a certain amount of albumin with hyaline or granular casts.

In intensely infective cases, acute nephritis may occur. Examination of the blood reveals moderate grades of leukocytosis.

In cases of diphtheritic enteritis secondary to infective lesions elsewhere, high fever, intense intestinal symptoms, and rapid prostration of strength are usually encountered. The evacuations in these cases may contain shreds of pseudomembrane or of exfoliated mucosa, and sometimes blood and pus. Other evidences of general infection are also met with.

Cholera Nostras.—Cholera nostras, or cholera morbus is an intense form of acute enteritis, bearing the same relation to ordinary cases of enteritis of adults as cholera infantum bears to ordinary enterocolitis in childhood.

Etiology.—This form of enteritis occurs with greatest frequency in the summer months, and is usually, although not always, traceable to some distinct error of diet. The eating of fish, shell-fish, unripe or tainted fruit, or a mixture of various indigestible substances, combined with the drinking of beer or other fermentable liquids, frequently occasions the disease. Probably in many cases bacterial infection plays an important part in the etiology, although no particular form of organism is associated with it. In some cases, preformed products of bacterial decomposition in the food are active; in other cases it is probable that, as the result of digestive disturbance after errors in diet, bacterial decomposition takes place in the stomach or intestinal tract.

Chilling is an important etiological factor. Exposure during cool nights following hot days, and when the surface has been relaxed and moistened by active perspiration, is particularly important.

The pathological anatomy does not differ essentially from that of other forms of enteritis. There is, however, a tendency to marked exfoliation of the mucous membrane and follicular infiltration; and, as the consequence of intense serous outpourings, desiccation and anæmia of various organs may be discovered.

Symptoms.—The onset is sudden and violent, usually beginning with nausea and intense vomiting and violent abdominal pain. The patient speedily falls into a condition of prostration, from the intensity of the suffering and the violent nausea and vomiting. The contents of the stomach are soon evacuated, after which bilious vomiting takes place; and finally, watery liquid is discharged, or intense unproductive retching may ensue.

Soon after the onset diarrhœa begins. After the discharge of the contents of the lower bowel the movements become copious, watery, and frequently repeated. They are discharged without special effort or tenesmus, and may finally become almost or quite involuntary. From beginning to end the tendency to large, watery evacuations is a marked feature.

The abdomen is tender, and sometimes excessively so. The patient in the beginning lies upon the side, with the legs flexed to relieve pressure, or upon the back, with the thighs flexed upon the body. Later, sensation

becomes less acute, owing to prostration, and pain and tenderness become less marked.

At the onset the temperature rises, and sometimes reaches a considerable degree of elevation. Exceptionally, hyperpyrexia may occur. Later, as prostration increases, the surface temperature falls; and finally, the extremities, the face, or the whole body may become cold. The rectal temperature, however, may remain elevated to the end. The secretions, and especially the urine, become scanty. Albumin and casts are generally present.

In severe cases the patient falls into a condition of collapse, hardly distinguishable from that of true Asiatic cholera. The face is cold and cyanosed, the lips dry, the tongue parched, the breath cold, respirations feeble, the voice suppressed, the pulse weak and thready, and the general condition is one of intense prostration, stupor, or collapse.

In mild cases, after a sudden onset and somewhat intense symptoms, the diarrhœic discharges become less severe, the vomiting ceases, fever subsides, and a gradual amelioration of other symptoms occurs. After a day or two practical recovery may ensue. In severe cases the symptoms continue unabated or increase in severity, and finally collapse occurs.

Diagnosis.—The recognition of enteritis offers no great difficulty in most cases. The history, onset, abdominal pain, and diarrhœa are characteristic. When the large bowel is considerably involved it may be difficult to determine whether the enteritis is one of ordinary type or dysenteric. Bacteriological examination of the stools and serum reactions may in these cases be required to establish a correct diagnosis. Sometimes enteritis presents a puzzling resemblance to typhoid fever. The suggestive features in these cases are continued fever, diarrhœa, general prostration, loss of appetite, coated tongue, and headache. The occurrence of splenic enlargement, rose spots, and the characteristic serum reaction eventually establishes the diagnosis in cases of typhoid fever, but a week usually elapses before these indications occur. Before that time leukocytosis, negative blood culture, general abdominal soreness, and the urgency of the diarrhœa may aid in excluding typhoid fever.

✓ Allusion has been made to the difficulty of distinguishing some cases of enteritis from appendicitis. As a rule, the correct interpretation will be made possible by the persistence and increase of the local signs (tenderness, rigidity, tumor), by the occurrence of repeated nausea and vomiting and perhaps chills, and by increasing leukocytosis. Obstinate constipation, while not necessarily diagnostic, is strongly suggestive of appendicitis.

The diagnosis of cholera nostras usually offers no difficulty whatever. In times of epidemics of Asiatic cholera, however, cases occur in which the diagnosis cannot be made. Under these circumstances no safe rule can be adopted other than to regard all choleraic diarrhœas as of the specific type, particularly as it is well known that mild cases of true Asiatic cholera are frequently seen in times of epidemic prevalence of that disease.

Prognosis.—The prognosis in cases of ordinary enteritis in adults is favorable. Unless the cause is one of unusual severity, recovery usually takes place. In the old or enfeebled, however, as in children, the disease is one of considerable gravity. Usually after a few days of urgent symptoms, gradual amelioration takes place, terminating in complete recovery. Sometimes the condition ends in a subacute, or even a chronic, intestinal disease.

Treatment of Acute Enteritis.—Prophylactic treatment is of the first importance. Among important preventive measures are the avoidance of irritants, tainted food, and the like, especially in hot seasons; in institutions in which numbers of persons are closely associated, or in camps, barracks, and the like, the closest scrutiny of the food and water supply during the hot seasons of the year is necessary. Careful attention to the dress, especially the wearing of abdominal bands, is of importance in susceptible individuals, and is a useful precaution that is commonly observed in tropical countries.

The treatment of acute enteritis is practically the same as that of ordinary diarrhoea, and therefore needs no extended discussion here.

The patient should be kept strictly at rest. The temptation to remain out of bed occurs only in mild cases, as the symptoms in severe forms are usually marked enough to make the patient take to bed at once. In the beginning of mild cases, however, matters may be made more serious by any attempt to continue at ordinary occupations, and in severe cases premature release from the bed may cause recurrences.

Warm applications, such as fomentations, hot-water bags, poultices, or the like, not only give great comfort but have a controlling effect on peristalsis, and thus aid in checking diarrhoea. When distention of the abdomen is troublesome, turpentine stupes may be advantageous.

As soon as the nature of the disease is recognized, the first care must be the removal of irritants from the intestinal tract. Unless diarrhoea is active from the first, a full dose of castor oil, rhubarb, or some saline may be used to evacuate the contents of the bowel. If the symptoms in the beginning are not urgent, calomel followed by a saline may be employed.

The diet should be restricted to liquids, such as boiled milk, broths, gruels, albumin water, and the like, or should be interdicted entirely. When vomiting or gastric symptoms are present, it is, as a rule, advisable to withhold all food in the beginning.

After copious evacuation has been secured or has occurred spontaneously, measures should be taken to control the diarrhoea and the intestinal inflammation. Bismuth salts, chalk mixture, lime-water, and other alkalies are advisable when acid fermentation is active. Astringents, such as kino, catechu, hæmatoxylon, or various other tannic acid preparations may be used to control diarrhoea. The addition of opium in the form of Dover's powder, laudanum, or paregoric is advisable when diarrhoea is severe and pain is marked. A timely injection of morphine in the beginning may control the severity of the attack. In later stages, when the diarrhoea has become less urgent and the general symptoms are less pronounced, such astringents as silver nitrate, acetate of lead, or sulphate of copper may be used in combination with opium.

In cases of cholera morbus the prompt use of opium by mouth or rectum, or of hypodermics of morphine, is advisable. The intensity of the vomiting usually precludes the administration of remedies by the mouth until the condition has been somewhat controlled. After this result has been secured, the treatment may be conducted as in ordinary cases.

When symptoms of collapse appear, hot applications over the abdomen are advisable; hot drinks, with the addition of small doses of brandy or other stimulants, may be given; and hypodermic injections of strychnine, camphorated oil, tincture of digitalis or digitaline, etc., should be employed.

When the symptoms have somewhat subsided, great care should be exer-

cised in resuming ordinary diet. Small quantities of liquid food alone should be permitted, and these should be given at short intervals and cautiously increased. If a tendency to nausea persists, cerium oxalate with fractional doses of cocaine or codeia may be administered before food, so that the stomach may be less irritable.

CHRONIC ENTERITIS.

Chronic enteritis is a condition of catarrhal inflammation of the small intestine and the upper part of the large intestine, with or without ulceration. It usually follows as the sequel of acute attacks.

Etiology.—This disease may result from a severe attack of acute enteritis or may be caused by repeated slight attacks. Not infrequently it is an accompaniment of other conditions of the bowel, as is indicated in the sections on carcinoma, intestinal obstruction, faecal impaction, etc. Certain individuals show a distinct tendency to repeated or chronic catarrh of the bowel.

Pathology.—The intestines usually present evidences of long-continued inflammation of the mucous membrane. The surface is covered with excessive secretion (mucous or mucopurulent), and there are areas of thickening of the mucosa or of erosion and atrophy. Ulcerations and stellate cicatrices of healed ulcers may be found, especially in the large bowel. Sometimes polypoid formations are found surrounding ulcers or independent of any ulceration. The mucosa may be of a normal color or may be hyperæmic and more or less pigmented. When distention of the bowels has been persistent, a thinning of the mucous membrane from overstretching may occur.

Symptoms.—The conspicuous symptom of chronic enteritis is persistent or intermittent diarrhœa. The movements are extremely variable in character, sometimes being nearly normal, at other times soft and pultaceous, or foamy, from the occurrence of fermentative processes. In still other cases they are large and liquid, and their color varies from a light yellow to a dark brown. Frequently they become highly offensive during exacerbations, when increased bacterial putrefaction occurs.

In some cases, instead of diarrhœa, there is persistent constipation. In these cases light-colored or gray scybalous masses, more or less coated with mucus may be passed; while at intervals, attacks of diarrhœa, with soft or liquid movements, occur. When the large intestine is especially involved, mucus is always a pronounced feature in the movements, and at times masses of mucus may be passed without any faecal matter. Blood-streaked movements or decided losses of blood may occur in cases attended with ulcerations or polypoid conditions of the mucosa.

The abdomen is usually distended when chronic enteritis affects a large part of the intestinal tract. In cases in which the process has become limited to a restricted area, distention may be wanting, and local discomfort or tenderness alone may be discovered.

The general condition of the patient suffering with chronic enteritis varies greatly. In many individuals this condition persists for years without great impairment of the health. In others, emaciation, general depression, and a variety of nervous symptoms significant of a lowered state of vitality

are met with. Hypochondriac and neurasthenic symptoms are not unusual, as in cases of mucous colitis. The patient may become extremely emaciated, anæmic, and weakened, and not rarely sinks into a state of semi-invalidism. The highest grades of anæmia are observed in cases in which associated ulceration in the colon causes loss of blood or in those in which bacterial putrefaction occurs actively.

Diagnosis.—It is difficult to distinguish chronic enteritis from functional disturbances of the intestine on the one hand, and from mucous colitis or certain primary nervous disorders on the other. In enteritis affecting the upper bowel the stools are found to contain undigested food, and are often acid in reaction, as in cases of intestinal indigestion. The intensity of the symptoms and the associated indications of inflammatory trouble alone make a distinction possible. No absolute line of division can, however, be drawn between the two conditions.

When the large intestine is especially involved, and mucus is abundant in the stools, chronic enteritis closely resembles mucous colitis. The latter however, is usually so closely associated with primary conditions of nervous debility, and the evidences of intestinal irritation are so inconspicuous, as compared with those in enteritis, that a distinction is usually easily made. The history of onset is also of importance in establishing the diagnosis.

Many individuals suffering with nervous diseases, such as neurasthenia, are liable to attacks of diarrhœa, or may suffer with more or less continued looseness of the bowels, which may suggest an intestinal origin for the nervous condition. In these cases, however, acute symptoms of irritation of the bowels are generally wanting and the manifestations are those of intestinal indigestion, rather than of enteritis.

When a chronic form of enteritis occurs in tuberculous or syphilitic persons it may be difficult to determine whether it is a simple enteritis or one of specific character. The clinical course and the results of treatment must determine.

Prognosis.—Chronic enteritis may continue for weeks, months, or years. The prognosis is always uncertain; but persistent treatment is usually partially, if not completely, effective.

Treatment.—The removal of all sources of irritation is the first care in the treatment of this condition. Sometimes an established error of diet may be discovered and corrected. In other cases a modification of the habits of life and the avoidance of exposure, fatigue, and similar causes are important. A close supervision of the patient's diet and an individual study may discover idiosyncrasies of fundamental importance.

Sometimes the correction of gastric digestion and the use of remedies such as *nux vomica* or other stomachics and digestive ferments may relieve the condition. Usually, however, some form of astringent medication is indicated. Of all remedies of this class, nitrate of silver is the most important. This may be combined with extract of opium or extract of *nux vomica*, and administered in the form of pills coated with salol or keratin, which secure the passage of the pill through the stomach and its solution in the alkaline secretions of the intestine. Other astringents, such as acetate of lead, sulphate of zinc or copper, and bismuth salts, may also be employed. All the remedies are best given in small doses frequently repeated. When fermentative processes are active, guaiacol carbonate, salol, and others of the so-called intestinal antiseptics may be employed. These also should be used in small

doses, as has been indicated by the experiments of Steele, who found that such remedies, by irritating the mucous membrane, may increase rather than decrease bacterial proliferation.

In cases involving the large intestine local treatment is advisable, such as repeated irrigations of the colon with water or saline solutions. When mucous formation is abundant, astringent injections, as solutions of nitrate of silver (1 to 5000), quinine (1 to 5000), or the addition of fluid extract of hamamelis to a saline solution (1 to 125), may be employed.

COLITIS.

Inflammation of the colon frequently occurs as an accompaniment of general catarrhal inflammation of the bowels or enteritis, and is a common accompaniment of organic diseases of the large bowel, such as carcinoma, strictures, faecal impaction, and the like. It is also met with as a manifestation of certain specific infections, such as dysentery. Occasionally, however, colitis occurs as an independent disease. This form alone is now under consideration. The term *simple colitis* has been applied to such cases by Hale White.

Etiology.—The causes of colitis are similar to those of enteritis, and no adequate explanation can be given for the limitation of the disease to the large bowel. Some cases seem to follow the dislodgement of hardened masses of faecal matter from the sigmoid flexure. Doubtless the retention of scybala in the pouches of the large bowel is quite sufficient to cause bowel inflammation. Dietary errors and various irritants doubtless play a part, and often infections are concerned. No specific microorganism, however, is operative in these cases.

Colitis sometimes occurs as a secondary manifestation in certain general infectious diseases, such as pneumonia, septicopyæmia, etc. It may also occur in cases of nephritis and uræmia.

Pathological Anatomy.—The mucous membrane of the bowel presents the usual appearances of inflammation such as have been described in the case of acute and chronic enteritis. Thickening of the mucosa, excessive mucous formation, and follicular ulceration are observed. In persistent cases polypoid formations may be met with, and pigmentation or hemorrhagic extravasation may be seen. In the colitis attending violent infections pseudomembranes are sometimes observed.

Symptoms.—The principal manifestations are diarrhoea, local pain, and tenderness. The stools contain an abundance of mucus, and are often blood tinged. Sometimes considerable hemorrhage may occur. In severe cases, small mucous, blood-tinged discharges, resembling those of simple dysentery, occur, and may be frequently repeated. In subacute cases faecal matter, with mucus and blood, is passed. Abdominal pain, especially in the region of the sigmoid flexure, is usually complained of, and sometimes there is extreme tenderness on pressure. This may be so great that the patient lies with the left thigh flexed; and even the weight of the bed-clothes may be intolerable. The pain is increased at the time of the movements, and sometimes active or violent tenesmus may occur.

Fever and general prostration of more or less intensity are usually present, and gastric disturbances may occur, although these are not, as a rule, severe.

In old and debilitated persons extreme prostration frequently presents itself. In the pseudomembranous colitis accompanying infections a rapid loss of strength, high fever, and prostration result from the intestinal complication.

Diagnosis.—The acute onset, local tenderness, and mucous discharges are the most characteristic features. It is extremely difficult, however, to distinguish a simple colitis from dysentery or from enterocolitis with especial involvement of the large bowel. It must also be remembered, in reaching a diagnosis, that colitis occurs as a symptom of malignant disease, strictures, and other organic conditions of the intestine.

Prognosis.—This is usually favorable, although a tendency to chronic inflammation sometimes results. In persons weakened by age or previous disease fatal terminations may occur.

Treatment.—Rest and warm applications to the abdomen, with careful regulation of the diet and the administration of remedies to check intestinal irritation, are the important features of treatment. All of these have, however, been sufficiently considered in the discussion of the treatment of diarrhœa and enteritis. Special mention may, however, be made here of certain local methods of treatment. When the inflammation in the sigmoid flexure and rectum is pronounced, and tenesmus is a troublesome symptom, ice suppositories, injections of small quantities of cold water, or of starch water containing opium, cocaine, or other sedatives, may be used with advantage. Cases marked by more extensive irritation, or in which hard scybala tend to form, are benefited by repeated injections of small quantities of olive oil. In subacute cases and during the later stages of acute attacks astringents of various sorts may be used in larger colonic irrigations. (See Enteritis.)

DIARRHŒA IN CHILDHOOD.

The diarrhœas of infancy and early childhood require especial consideration, on account of their greater urgency and their distinctive features. As the digestive and metabolic functions are relatively predominant at this age, any derangement of the gastro-intestinal tract naturally involves a greater disturbance of health than at other periods of life. Errors of diet and their consequences are, therefore, most serious in infancy and childhood.

Etiology.—The causes of diarrhœa in children are practically the same as those that operate in adults, but the difference in the character of the food, in the routine of life, and in susceptibility give a greater prominence to certain conditions.

Food.—Probably the most generally active cause of diarrhœa in children is improper feeding. A mere excess of food may be sufficient to derange digestion and occasion intestinal irritation; but overfeeding is particularly harmful because it furnishes favorable conditions for bacterial growth and fermentative processes in the intestinal tract. This is seen with greatest distinctness in young children past the nursing age, in whom a single overloading of the stomach may occasion an attack of diarrhœa that is afterward prolonged by conditions that arise secondarily. The more indigestible the food the more promptly will diarrhœa follow and the more serious its type. In sturdy, bottle-fed infants an appetite somewhat too keen may readily tempt the mother or nurse to overfeed the child, and diarrhœa frequently results. The quality of food, especially milk, must be considered.

Diarrhœa in young infants is frequently caused by milk containing too high a percentage of fat.

Much more commonly the occasion of the diarrhœa in childhood is some qualitative changes in the food, especially the use of tainted milk. Improved conditions in dairies and improved methods of keeping and transporting milk have materially lessened the frequency of the diarrhœas of infants; but, despite all efforts, diarrhœa due to bad milk is still the most frequent of all forms. Attempts to improve conditions by pasteurization or sterilization of the milk are of questionable utility, although the former plan is greatly preferable. Sterilized milk should be condemned, because its constant or repeated use undoubtedly occasions nutritional disturbances, infantile scurvy, and other diseases. Pasteurization, although less objectionable, cannot be relied upon to destroy all microorganisms, and thus often gives a false sense of security. Experimental investigations have, moreover, shown that after thorough pasteurization, bacterial multiplication occurs even more rapidly than in raw milk; and when the food is prepared in the morning, the bottles given later in the day may be in worse condition than if pasteurization had not been practised. Pasteurization of the milk on a still larger scale, as when it is carried out by the dairyman or supplier, is still more objectionable. The addition of any bactericidal agent to milk, even in minimal quantities, must be unreservedly condemned. Intense forms of diarrhœa and general infection are sometimes followed by the use of milk obtained from cows suffering with mastitis. This condition is caused by a streptococcus that produces virulent infection.

Safety in milk feeding lies in the careful supervision of dairies, the handling of the milk in the cleanest possible manner, prompt delivery at the home, and careful refrigeration after its delivery.

Sometimes, although the milk used has been fresh and pure, diarrhœa may follow its use, on account of disturbance of digestion and secondary fermentative processes caused by bacteria already present in the intestinal tract. Often this is caused by the use of milk mixtures that are too strong and therefore indigestible. Every attempt at correcting such diarrhœa by modifications of the milk, pasteurization, suitable dilutions, etc., may in some cases be wholly futile.

Diarrhœas due to milk may be the result of the action of bacteria present in the milk, or may more directly result from poisoning by bacterial products present in the milk. The latter forms are particularly severe, and more properly merit the term milk poisoning, which is often used in a general sense to indicate diarrhœal disease due to milk feeding.

Recent investigations have shown the presence of an organism closely allied with the *Bacillus dysenteriae* of Shiga in cases of infantile summer diarrhœa of the enterocolitis type. There is little doubt that this organism plays an important part in the etiology of some cases and of occasional extensive outbreaks in institutions or in certain communities. It is quite as certain, however, that cases or outbreaks clinically indistinguishable from these are caused by other microorganisms. Among these are various closely allied organisms differing mainly in their capacity to ferment different forms of sugar. Other organisms, however, such as the *Bacillus lactis aërogenes*, the *Bacillus coli* and close allies of this group, streptococci and staphylococci are concerned in some cases. In many cases several forms seem to be associated.

The most intense diarrhœas of the cholera infantum type seem to be caused by products of bacterial decomposition either occurring in the food or formed in the intestinal tract. No special microorganism has been connected with these cases.

In older children, also, various other foods may be the cause of attacks of diarrhœa. Unripe or spoiled fruit, particularly, takes high rank. In certain epidemics it has seemed probable that the source of infection was water. The careless use of impure water in the washing of milk cans or bottles may cause infection of the milk, or the drinking water given the child may cause direct infection.

A variety of causes affecting the susceptibility of the child and the liability to food infection will now require consideration.

Age.—The time of greatest susceptibility is from the sixth to the twentieth month, the period of the primary dentition. Dentition itself, by causing reflex disturbances, excessive secretions, or fever, increases the liability to all forms of diarrhœa, although it may not be the immediate, direct cause.

Season.—Diarrhœa is distinctly a disease of warm weather, although it may occur at any period of the year. In the summer months it is always more difficult to preserve milk and other foods. In addition, the children are rendered susceptible by free action of the skin and chilling at night.

Environment.—Surroundings play an important role in the causation. Children in crowded cities and amid surroundings of destitution and squalor are much more liable to severe diarrhœas than those in the country or living under more wholesome conditions. In institutions in which a number of children are in close contact direct infections frequently occur.

The writer has repeatedly seen, in the babies' ward of a children's hospital, one child after another attacked with diarrhœa after the admission of a case or the development of one in the ward. The greatest care is necessary, in the handling of children, to avoid the communication of diarrhœal conditions; particularly should the handling of the diapers be scrutinized with care. They should never be kept in the room, and should be thoroughly sterilized before they are used again. Food standing in the same room may easily become infected, and the preparation of the food should be carried out in a well-ventilated apartment, away from any danger of contamination from bad drainage, sewage arrangements, ventilators, etc.

Pathology.—The lesions in the various forms of infantile diarrhœa vary considerably; but quite commonly the character of the anatomical manifestations is not in keeping with the seriousness of the symptom.

Three types may be distinguished, although no sharp dividing line can be drawn between them. These are: (1) simple or dyspeptic diarrhœa; (2) enterocolitis; and (3) choleraic diarrhœa or cholera infantum.

In the first of these the bowel may present no evidences of disease post-mortem. When the manifestations have been severe, some indication of catarrhal irritation or inflammation may be found in the upper small intestine and stomach. Congestion, slight thickening, and excessive mucus formation may be discovered.

In the enterocolitis of childhood more definite changes in the mucous membrane are seen. Redness and swelling, and frequently enlargement of the lymphatic follicles, are usually met with. Sometimes small follicular ulcerations, and less commonly swelling and slight ulceration of Peyer's patches, may be seen, especially in subacute or chronic cases. In cases

of excessive severity pseudomembranes or extensive ulceration may occur, more particularly in the large bowel. After long-continued attacks of enterocolitis, and those in particular that are attended with continued distention, a thinned, atrophic mucous membrane may be found. Various secondary changes in other organs expressive of depletion of the system occur, as in other diarrhoeal affections. Infective lesions, such as nephritis and degenerations of the liver, and other glandular organs, are among the occasional pathological features.

In cholera infantum evidences of intense inflammations of the mucous membrane, with exfoliation of epithelium and swelling of the glands and lymphatic follicles, may be seen.

Symptoms.—**Simple or Dyspeptic Diarrhoea.**—Usually evidences of gastric disturbance precede the attack of diarrhoea, and attend it more or less continuously. The child regurgitates food or is attacked by active vomiting. At the same time he becomes restless and distressed, with abdominal pain and distention. The facies and actions of the child suggest griping abdominal pain. Later, diarrhoea sets in, the movements being offensive or sour-smelling, and characteristically containing undigested food, such as curds of milk, together with more or less mucus. The color of the stools varies greatly, sometimes being white or light colored, from the presence of large amounts of undigested milk and unabsorbed fat; at other times, green, from the presence of biliverdin or of coloring matters produced by bacterial action. Lesage, in particular, has shown that chromogenic bacteria are active in producing green stools in certain cases. The number of movements may be merely a slight increase over the normal, or may reach ten or more in the twenty-four hours.

In some cases the evacuation of the bowels is closely dependent upon the taking of food, one or more movements quickly following each feeding. In such cases there is apt to be a considerable amount of undigested food in the motions.

The general condition of the child is one of moderate prostration, although frequently there is but little weakness until the diarrhoea has lasted for some time. If long continued, emaciation, pallor, and other signs of depletion are met with.

Enterocolitis.—The term inflammatory diarrhoea is also applied to this variety. The symptoms of this form of diarrhoea are distinctly more intense, and in marked cases reach high grades of severity. Nearly always the child loses strength rapidly, and when repeated, and especially watery, stools are discharged, extreme prostration may speedily occur. Fever is practically always noted, and in severe cases is very high. Temperatures ranging from 103° to 105° are not unusual, and hyperpyrexia is a common manifestation of the enterocolitis of the hot summer season.

The onset of the disease is usually abrupt, and is attended with evidences of general distress, and especially abdominal pain. Vomiting frequently occurs, but is less conspicuous than in the dyspeptic form, and sometimes, especially in older children, may be wanting. Often, however, obstinate vomiting is a marked symptom.

The movements are numerous, and vary in color from brown to green. After the contents of the bowel have been evacuated, more watery movements, with less and less color, may occur, so that the condition approaches, in the character of the discharges, to the choleraic type. When the large

bowel is especially involved, considerable mucus may be mixed with the discharges.

The general condition of the child is one of rapid exhaustion. The face becomes pinched, the eyes grow hollow and are surrounded by circles, the temples are sunken, and when severe exhaustion has developed, the hands and feet may grow cold and cyanosed, while the internal temperature as determined in the rectum remains excessively high. The abdomen is often distended, and the skin tensely drawn and harsh. The child becomes more and more feeble, its movements less active, its cry more and more faint, and its lips dry. Finally, it sinks into a state of complete collapse. An apathetic, stuporous, or even comatose condition, now and then interrupted by a complaining cry, betokens the development of a condition resembling that of meningitis, and designated as spurious hydrocephalus.

In cases involving the large intestine to a considerable degree, violent straining occurs with each evacuation of the bowels, and prolapse of the rectum is a not unusual feature. In all cases the bowel movements may be irritating and cause inflammations of the skin about the anus or buttocks.

The urine early becomes scanty, and occasionally the secretion is almost suppressed. Superficial œdema may result from this interference with the renal function. Actual nephritis occurs in the intensely infective cases.

When the temperature remains persistently high, various nervous symptoms, such as restlessness, slight twitching, and even convulsions, may occur; but the tendency to prostration usually supplants all other nervous indications, and the child sinks into the condition of lethargy or coma already referred to.

The loss of flesh and strength sometimes increases with startling rapidity. Within a few hours the child may be visibly reduced, and when the condition has lasted for several days extreme grades of desiccation and inanition result.

Complications, such as bronchopneumonia, hyperpyrexia, and extreme nervous symptoms, often precede the fatal termination, but more commonly this results directly from exhaustion.

DIAGNOSIS.—The recognition of this form of diarrhœa offers little difficulty. In the beginning, high fever and the absence of characteristic intestinal manifestations may suggest infectious diseases of various sorts, but usually the characteristic symptoms speedily present themselves. It is practically impossible to separate cases due to ordinary causes from instances of epidemic diarrhœas produced by the *Bacillus dysenteriae*, except by bacteriological methods.

PROGNOSIS.—Enterocolitis is always a serious disease in childhood, particularly in young infants. Older children more commonly exhibit enough resistance to withstand the attacks.

Choleraic Diarrhœa, or Cholera Infantum.—This form of disease, usually met with in early infancy, is of the most extreme gravity. The onset is sudden, and the child becomes prostrated very early. After a few movements, in which the bowel contents are evacuated, a choleraic form of diarrhœa occurs, the stools being large and watery, and containing but little faecal matter.

The whole body undergoes a rapid shrinking, and the features present the shrunken, hollow appearance described as occurring in the most violent cases of enterocolitis, but coming on with even greater rapidity. Extreme dryness of the tongue and mouth and excessive thirst are evidences of

the desiccation. The urine is scanty or suppressed. The blood becomes inspissated, and the circulation is interfered with, so that cyanosis of the hands and feet, or of the whole surface, becomes marked. The eyes sink in their sockets, the fontanelle is depressed, and within a few hours the child's face assumes the wrinkled appearance of senility. The surface is cold, and stupor or coma develops rapidly. The surface temperature is greatly reduced, while the internal temperature, as observed in the rectum, may rise to 105° or more.

The course of this form of diarrhœa is brief, and in a large proportion of cases terminates fatally. When improvement occurs there is a subsidence of temperature, a gradual cessation of diarrhœa, and a slow restoration of strength. Not rarely a less degree of diarrhœa persists for some time, due to a residual enterocolitis.

PROGNOSIS.—The mortality from this form of diarrhœa is exceedingly great. When it occurs in institutions containing large numbers of infants, a fatal termination may take place in above 90 per cent. In older children the outlook is less grave, although it is always a condition of extreme severity.

Treatment.—The prophylactic treatment of infantile diarrhœas is of the greatest importance. Greater care in the preparation and preservation of food and increased knowledge of the causes of diarrhœa have led to a decided diminution in the frequency of these conditions, although much still remains to be accomplished.

In hot seasons of the year all forms of food, but particularly milk, should be kept with the strictest care. Fortunately, municipal and voluntary associations, such as milk commissions, have done much toward correcting many of the abuses that formerly existed in the matter of the production and distribution of milk. When children are bottle-fed, the food if prepared in the morning for the whole day should be kept in a refrigerator. Careful pasteurization is often necessary when the freshness of the milk is doubtful. In the early morning some other form of food, such as barley water or prepared foods, may be used with advantage until the new supply of the day has been received. In the cases of older children rigid care should be taken to avoid the use of stale foods of all sorts.

The susceptibility of the child may be lessened by the avoidance of exposure. In the summer, when the nights are relatively cool, care in the dress is of importance, particularly the protection of the abdomen and legs.

Infectious diarrhœas may be avoided in institutions by the separate management of the children and by the closest care in the handling of the napkins.

Medicinal Treatment.—After the onset of diarrhœa, treatment should be directed to the removal of irritants, the control of the diarrhœa, and the support of the patient's strength.

In the milder dyspeptic diarrhœas the prompt administration of a dose of castor oil and the withdrawal of the accustomed food, substituting for this barley water, rice water, albumin water, weak broths and the like, may speedily terminate the condition. If the tendency to diarrhœa persists, mild astringents, such as powders of bismuth subnitrate, subgallate, or subcarbonate, with or without the addition of pepsin, may be sufficient. More active astringents, like chalk mixture with kino or catechu, and tannic

acid preparations of other sorts, may be used. Frequently, small doses of calomel, alternating with bismuth, chalk mixture, or other astringents, appear to have good effect. An old method of treatment was the administration of rhubarb with paregoric or laudanum. When the movements are acid and irritating, alkalies, such as magnesia, soda, or lime-water, may be employed.

In cases of enterocolitis, preliminary purgation may be desirable, although it must be used with greater caution than in the dyspeptic diarrhœa, because the tendency to spontaneous evacuation of the irritating matters in the bowel is greater and prostration is more imminent. Sometimes, however, the prompt administration of castor oil, rhubarb, or magnesia is desirable. When vomiting is severe, all food should for a time be withheld, or only diluted barley water, rice water, or thin gruel should be given. At the same time, small doses of calomel, alternating with bismuth powder, may be employed. In later stages, astringents, such as have been mentioned before, are useful. Opium in the form of laudanum, Dover's powder, or paregoric is often necessary to check obstinate diarrhœa; and sometimes, when violent purgation threatens speedy collapse, a hypodermic injection of morphine (gr. $\frac{1}{30}$ to gr. $\frac{1}{20}$) may exercise a rapid controlling effect.

When the temperature is high and prostration is severe, colonic irrigations are most serviceable. By gentle manipulation, with the hips elevated, the colon may be flushed without exciting irritation of the bowel, and the effect upon the general condition of the child is frequently surprising. The water used for irrigations should be of a temperature of from 90° down to 80° or 75° F.; the amount may vary, according to the age of the child, from half a pint to a quart. A catheter or small-sized rectal tube, passed high into the bowel, and a fountain syringe, elevated not above a foot or two, suffice for the injections. If the colon and rectum are irritable, the first injection may be at a higher temperature; and a few drops of laudanum may, with advantage, be added to the fluid. Sometimes a small amount of oil may first be injected; or a little starch water, containing laudanum, cocaine, or other sedatives. In later stages, if colitis persists and slight diarrhœa with abundant mucus continues, injections of fluid extract of hamamelis in normal saline solution (1 to 65) or nitrate of silver (1 to 8000) may be employed. Stimulants and other supporting measures may be necessary on account of threatened collapse. Hypodermics of strychnine (gr. $\frac{1}{200}$ to gr. $\frac{1}{400}$) and small doses of brandy, tincture of digitalis, or tincture of nux vomica may be used. If the external temperature has remained high, sponging or cold packs may be employed with advantage.

In cholera infantum the tendency to speedy collapse requires the prompt administration of stimulants, such as those named. External heat may be needed when the surface temperature sinks, and injections of fluid under the skin may be necessary to counteract the loss of water from the system.

The diarrhœa itself may be controlled by the use of astringents administered as freely as the condition of the stomach permits. In many cases the prompt administration of a hypodermic injection of morphine will check vomiting and diarrhœa, and permit of the administration of other remedies.

If the diarrhœa is got under control the cautious increase of nourishment and the use of stimulants are required to restore the patient's strength. The greatest care must be taken, however, to prevent a recurrence of the intestinal disturbance.

Serum treatment has been employed since the discovery of organisms resembling the bacillus of Shiga in cases of infantile diarrhoea, but the results have not proven especially encouraging.

ULCERATION OF THE BOWEL.

A classification of intestinal ulceration is difficult, and from many stand-points unsatisfactory. The most rational presented is probably that of Nothnagel:

First Group.—Ulceration as the result of neerotic processes:

Simple duodenal ulcer (including peptic ulcer of the jejunum).

Ulcer following cutaneous burns.

Embolie and thrombotic ulcer (the peculiar ulcers of the intestine seen in patients with multiple neuritis belong to this class).

Amyloid ulcers.

Second Group.—Ulceration as the result of inflammatory processes:

Catarrhal ulcer.

Follicular ulcer.

Simple ulcerative colitis.

Stercoral or decubital ulcer.

Third Group.—Ulceration as the result of acute infectious diseases:

Typhoid.

Dysentery.

Diphtheria.

Anthrax.

Sepsis.

Erysipelas.

Varioloid.

Fourth Group.—Ulceration as the result of chronic infectious diseases:

Tuberculosis.

Syphilis.

Erysipelas.

Leprosy.

Pellagra.

Fifth Group.—Ulceration as the result of constitutional diseases:

Gout.

Scurvy.

Leukæmia.

Sixth Group.—Toxic forms of ulceration:

Uræmic ulcer.

Mercurial ulcer.

In the sections that follow, "simple duodenal ulcer" will be considered separately on account of its special etiology and its peculiar clinical features. The other forms of ulceration, whether occurring in the duodenum or elsewhere, are properly discussed in another section. Jejunal ulcer is, in an etiological sense, related to simple duodenal ulcer rather than to the other group, but for the sake of simplicity of description, its symptoms will be given with the latter group. The ulcerations of the intestine met with in some acute infections, Nothnagel's third group, are more appropriately considered in connection with the infectious diseases in which they occur. Of

the ulcerations included in the fourth group, the tuberculous and syphilitic will be separately described in this volume. The intestinal lesions of lepra and pellagra are more properly described in the discussion of these diseases, as are also the ulcer, occasionally met with in the diseases of the fifth group. A brief reference to leukæmic ulceration and to the rare form of trophic ulceration (which is not included in the classification) may be in place.

Duodenal Ulcer.¹—The duodenal mucous membrane is frequently the seat of ulceration resembling that found in the stomach, and known as gastric or peptic ulcer. Ulcers due to infectious diseases, such as tuberculosis and typhoid fever, and various other forms will be considered elsewhere. The form now under consideration is that which results from digestive action rather than from infectious, toxic, or inflammatory processes. It has been designated simple duodenal ulcer or peptic duodenal ulcer.

Occupation, habits, and environment seem to play no particular part in the etiology, except that conditions that tend toward irregularity in eating, and especially to the use of alcohol, may favor the development of the condition.

Etiology.—Duodenal ulcer is found associated with certain diseases, notably of the stomach. As to the association of gastric with duodenal ulceration, according to the Fenwicks, 1.7 per cent. of cases of gastric ulcer were accompanied by a lesion of the duodenum, but Mayo and Moynihan have found that half their cases of duodenal ulcer were associated with gastric ulceration. The clinical history of cases indicates that chronic gastritis with excess of acid secretion is a factor of considerable importance in the development of duodenal ulcer. Nearly all patients give a history of antecedent gastric disorders.

Duodenal ulceration appears to be associated with tuberculosis rather more frequently than it is found in persons free of this disease or suffering with other maladies. Ulceration of the duodenum has also been referred to as occurring in chronic nephritis. These ulcers, however, are somewhat different in their general character from ordinary duodenal ulcers, and will be considered elsewhere.

Extensive burns of the skin have been for a long time known to be associated with duodenal ulcers. These also differ in character and causation from true duodenal ulcers, and will be more appropriately considered by themselves.

Pathological Anatomy.—Duodenal ulcers, like those of the stomach, may be distinguished as acute or chronic. The former are circular and punched-out in appearance, and the floor and edges are congested and soft. The chronic form presents thickened, indurated, and sometimes undermined and irregular edges, and not infrequently has a sloping form, which gives the ulcer a funnel shape. Acute ulcers may be quite superficial, presenting themselves as mere erosions of the mucous membrane. The more chronic forms are deeper, extending to the submucous or even to the muscular coat, and occasionally penetrating through all the coats, when the liver, the head of the pancreas, or some other neighboring structure may form the floor of the ulceration, adhesions having prevented an open rupture into the abdominal cavity. The mucosa surrounding an ulcer, although usually normal, may

¹ Duodenal ulcer of the form here discussed, which anatomically comes under the heading of ulceration of the bowel, is closely associated with gastric ulcer as regards etiology and symptoms. It is therefore discussed both under Diseases of the Stomach and in this section.—EDITOR.

present evidences of irritative change, and is sometimes irregularly elevated or polypoid. Microscopically the floor and edges of the ulcer present necrotic changes, with some increase in connective tissue, but without the evidences of active inflammatory conditions, such as are found in ordinary ulcerations.

The situation of duodenal ulcers is usually in the first part of the duodenum, near to the pylorus. The majority of the ulcers in the first part of the duodenum are situated quite near the pylorus, and are deepest at the upper part where the gastric contents are projected with greatest force against the duodenal wall. The anterior wall of the duodenum seems to be affected more frequently than the posterior, although all observers do not agree upon this point.

Secondary Changes.—Duodenal, like gastric ulcers, may undergo a process of gradual cicatrization; but apparently this termination is less common than in the case of ulcers of the stomach. Chvostek insists that cicatrization is more common than has been generally believed. Perry and Shaw estimate it as occurring in 11 per cent. of all cases. When it occurs there may result a stenosis or deformation of the duodenum, similar to the stenoses produced by cicatrizing gastric ulcers. Progressive dilatation of the stomach with symptoms of food stagnation result from this condition. Cicatricial stenosis of the lower end of the duodenum or the parts adjacent to the papilla of Vater may occasion obstruction of the biliary and pancreatic ducts. Occasionally diverticula of the duodenum follow the cicatrization of an ulcer. Deep ulcers may cause severe hemorrhage, from involvement of the large arteries adjacent to the duodenal wall—the pancreaticoduodenal, the gastroduodenal, the pancreatic, etc. The erosion of such vessels is usually followed by severe and sometimes fatal hemorrhage. This termination has been noted by the Fenwicks in 35 per cent. of their cases.

Perforation of the duodenum occurs in about half the fatal cases. Ulcers upon the anterior wall are more apt to terminate in this way than are those upon the posterior wall, owing to the absence of adhesions to neighboring structures. When a perforation takes place without the previous formation of any adhesions the contents of the stomach and duodenum have ready access to the peritoneal cavity, and usually occupy the right side of this sac, extending along the colon toward the pelvis. When adhesions have formed, or when the perforation is minute, a localized abscess may develop. In such cases secondary perforation of the abscess into the liver, the stomach, other portions of the intestines, or the gall-bladder, or through the diaphragm into the pleura, the mediastinum, or the pericardial sac, may occur. In a few instances, rupture into the aorta, portal vein, vena cava, or gall-bladder has been recorded.

Symptoms.—In a large proportion of cases duodenal ulcers are chronic in course and the symptoms are slow in development. Occasionally acute ulceration occurs, and the symptoms make their appearance suddenly, intense pain and acute disturbance of digestion, followed by the rapid development of hemorrhage or perforation, being met with. In the great majority of cases, however, such acute onset is wanting, the symptoms being slow in development and without characteristic significance in the earlier stages. In these cases the patient suffers with gastric manifestations and resulting emaciation, loss of strength and appetite, and finally attacks of pain suggesting hyperacidity. In many instances the condition may be entirely latent until hemorrhage or perforation takes place; and cicatrices

of old duodenal ulcers may be found at autopsy in cases in which no evidences had been manifested during the life of the patient to indicate the existence of the disease. Other authors have estimated the proportion of latent cases as somewhat lower. In the cases in which symptoms have developed the most marked are pain, dyspeptic manifestations, and hemorrhage.

The pain of duodenal ulcer is frequently paroxysmal, like that of gastric ulcer. It may be of intense severity or merely a dull discomfort at certain periods after the ingestion of food. In other cases, whether severe or dull, it is more lasting, and sometimes a constant sense of burning or of sharp pain is experienced. The pain is situated to the right of the middle line, and usually a little above the umbilical level. It may radiate toward the right, or in other cases extends toward the left side. Sometimes it is described as having a deep-seated location, being rather unlike the characteristic pain of gastric ulcer in this particular. The time of its appearance varies. Sometimes it follows immediately after the taking of food, but more commonly it does not occur, or does not reach its acme, until two, three, or four hours after meals. The character of the food may have a certain relation to the occurrence and the intensity of the pain. Generally speaking, heavy meals occasion more severe pain; although it may be somewhat later in appearing. The drinking of copious draughts of water or the taking of other liquids, such as milk, beer, wine, etc., may relieve the paroxysm for a time.

Tenderness and rigidity of the abdominal muscles may be met with over the seat of duodenal ulcers, especially those situated anteriorly. Pressure usually increases the painful paroxysms, although not invariably.

Gastric symptoms are rarely wanting in typical cases. In a great many instances there is a history of long-standing gastric disturbance. The patient will state that he has gone from one form of diet to another, in the vain endeavor to relieve himself of recurring pain after the ingestion of food. The symptoms usually complained of are distention, acidity, the regurgitation of sour liquid, and especially dull or sharp pain after the taking of food. Loss of appetite may occur. In other cases there is an intense craving for food, which is controlled only by the pain that follows its ingestion. Vomiting sometimes occurs. The general condition of the patient is suggestive. He is emaciated, sallow, or anæmic in appearance, the latter being striking when a slow and continued oozing of blood has occurred; and frequently he presents a desiccated, shrivelled appearance. The bowels are usually constipated, but may be variable, short attacks of diarrhœa alternating with longer periods of constipation. Examination of the stomach contents may disclose a hyperacidity, although this is not so frequently the case as the symptoms would suggest.

Hemorrhage may occur from the stomach or from the bowel, or the blood may be evacuated in both ways. Oppenheimer found vomiting of blood in 8 cases, discharge of blood from the bowels in 10, and a combination of the two in 16. More frequently than severe hemorrhage there is slight oozing of blood, which may occasion a discoloration of the stools that is just sufficient to be observable; or there may be so little blood that it can be recognized only by means of chemical tests (occult blood). The continuous presence of small amounts of blood, when associated with suggestive symptoms, is a diagnostic indication of great value.

Perforation of a duodenal ulcer may occasion symptoms similar to those met with in the perforation of gastric ulcers. Usually the onset of this complication is attended with sudden prostration and pain. The patient may sink at once into a condition of shock or collapse; later he may revive and pain in the abdomen, especially toward the right side, tenderness, and distention, due to the development of peritonitis, may then set in. In a great many instances the diagnosis of appendicitis has been made, on account of the acuteness of onset and the tendency to right-sided situation of the symptoms.

Complications.—Among the complications of duodenal ulcer are those which result from extension of the ulcerative process through the duodenal wall, and those which are due to cicatrization of the ulcerated area.

In the former group, the most important condition is localized peritonitis. In the case of slowly growing ulcers, a certain amount of localized peritonitis causing adhesion with neighboring organs, is frequent. When these adhesions fasten the duodenum firmly to some solid structure and the ulceration penetrates to the serous coat, a localized abscess may occur; or a further penetration into the various structures with which the duodenum has become adherent may take place.

Among the results of cicatrization of the ulcer, the most important is partial stenosis of the duodenum, which, in turn, occasions an enlargement of the stomach. Occasionally, when the ulceration has been situated near the papilla of Vater, obstruction of the common bile duct may occasion chronic jaundice, or there may be an obstruction of the pancreatic ducts.

Carcinomatous change may take place secondarily in duodenal as in gastric ulcer, although this is less common. When such a change takes place, clinical indications of carcinoma may be recognized.

Diagnosis.—A positive diagnosis of duodenal ulcer is frequently impossible. In particular, the distinction from gastric ulcer cannot be made with certainty. This distinction, however, is of minor consequence, as an ulceration just within or just outside the pyloric ring is, to all intents and purposes, of the same consequence.

Long-continued or recurrent burning or aching pain, presenting itself at a certain time, usually from two and a half to four hours, after eating, and attended with evidences of chronic gastric disease, hyperacidity, and stagnation of stomach contents, is a suggestive combination of symptoms. When hæmatemesis or melæna is associated, or when small amounts of occult blood are constantly found in the stools, the diagnosis of ulceration near the pylorus becomes more certain. Chvostek suggests a test, which consists in the giving of a little wine when the pain following a meal has become marked. This causes immediate cessation of the pain, probably, as Chvostek holds, because the wine irritates the stomach and causes reflex closure of the pylorus, thus preventing the further discharge of acid stomach contents into the duodenum. When the test is applied after small meals the pain may be arrested permanently; after a large meal, temporarily. This test, however, is successful in only a small proportion of the cases in which it is employed.

Besides the test just given, other indications of duodenal as opposed to gastric ulcers are the deeper position of the pain, its situation to the right of the middle line, and its radiation toward the right and downward. In addition to these facts, the time of the occurrence of the pain may serve to indicate the probable diagnosis.

Sudden rupture of a duodenal ulcer, with resulting peritonitis, may be mistaken for appendicitis, intestinal obstruction, acute pancreatitis, and sometimes gallstone. The tendency of the pain and tenderness to extend toward the right, and thus to suggest appendicitis, should be especially borne in mind.

The withered, emaciated condition of a patient who has had long-standing ulceration of the duodenum may suggest the existence of carcinoma at the pylorus, particularly when pyloric spasm, constant or paroxysmal, has occasioned enlargement of the stomach and visible peristalsis. There may even be a palpable mass due to periduodenal adhesions and inflammation. In such cases a positive diagnosis is sometimes impossible.

Treatment.—The treatment of duodenal ulcer is practically identical with that of gastric ulcer. In acute cases, prolonged abstinence from feeding by the mouth is the most important element in the treatment. The patients can, without difficulty, abstain from all food for two or three weeks, some nourishment, perhaps, being derived from nutrient enemata, and water being administered by the bowel. Bismuth subnitrate, olive oil, or nitrate of silver may be given by the mouth for the purpose of neutralizing acidity, protecting the ulcerated surface, and through an astringent effect stimulating cicatrization. If bismuth is used and complete starvation is practised, small doses will suffice, gr. 10 to 15 (gm. 1) being administered several times daily in suspension. Olive oil, in doses of from 2 drams to a $\frac{1}{2}$ ounce or 1 ounce, may be given several times daily; and nitrate of silver, in solution in distilled water, or in the form of freshly made pills. The doses of this drug should be small, not exceeding gr. $\frac{1}{4}$ (gm. 0.016), and preferably administered a number of times daily, rather than large doses at longer intervals.

Occasional lavage of the stomach with plain water, or with water containing bicarbonate of soda or other alkalies, may have a temporarily useful effect; but, as a rule, lavage should be avoided.

In less acute cases a more prolonged treatment of the same sort, with very carefully moderated feeding by the mouth, may prove successful. As a rule, however, subacute and chronic ulcers of the duodenum tend to persist, and eventually will require surgical treatment. It is wiser, therefore, to resort to surgical measures as early in such cases as a positive diagnosis of the condition can be made. When perforation of a duodenal ulcer takes place, immediate surgical operation is imperative.

Duodenal Ulceration following Extensive Cutaneous Burns.—Since Curling, in 1842, first called attention to the definite relationship between cutaneous burns and duodenal ulceration, the subject has been of deep interest. Without doubt the primary element in fostering this interest is the fact that, despite numerous attempts, as yet no satisfactory explanation for the relationship of the two phenomena has been presented. Theories that would rationally explain the occurrence of intestinal ulceration after extensive burns have been propounded, but none of them satisfactorily explains the fact that the duodenum alone is usually the seat of the phenomenon. From this standpoint the theory proposed by Hunter is the most rational. He explains the condition by assuming that, following the burns, toxic substances are secreted with the bile, which, on coming in contact with the duodenal mucous membrane, induce ulceration. He found substantiation for his view in the experimental occurrence of duodenal ulceration after the subcutaneous injection of toluylenediamin into dogs. However,

Fenwick was able to produce the same lesions on repeating the experiment after ligation of the common bile duct. Another view is that the condition is a result of emboli localizing themselves in the duodenal vessels, thus lowering the nutrition of the parts involved and subjecting them to digestion by the gastric juices. Cooke offers the theory that the violent trauma to the skin destroys the antiferments present in the mucous cells, and as the digestive powers of the gastric juice are most potent in the duodenum, digestion of the mucous membrane occurs here. A discussion of the various theories that have been proposed has been presented by Bardeen.¹

The ulcers differ in many features from the peptic duodenal ulcers. There may be but one ulcer or there may be from three to six present. They are usually located in the inferior horizontal portion of the duodenum. They may be irregular in outline or, as is usually the case, are long and narrow, varying in width from 1 to 5 mm. and in length from 5 to 15 mm. In exceptional cases, ulcers in the stomach or the lower part of the intestinal tract have been found associated with such duodenal ulcers. Usually the ulcer develops from the sixth to the twelfth day after the burn, but they have occurred as early as the second day and as late as the seventeenth. They are met with chiefly in young subjects, rather oftener in females than in males and more frequently after burns of the trunk than of the extremities. They are almost invariably fatal. According to the Fenwicks they occur in 6.2 per cent. of all fatal burns.

Jejunal Peptic Ulcer.—Jejunal peptic ulcer occurs after gastrojejunostomy from the same causes that induce duodenal peptic ulcer. It occurs just beyond the point of attachment of the jejunum to the stomach. It has been produced experimentally in animals by Watts. For some unexplained reason this condition is met with much more frequently in males than in females. Of 31 cases collected by Gorset, 29 were males. Jejunal ulcer results more commonly from anterior than from posterior gastrojejunostomy. This probably results from the fact that the motor activity of the stomach is less impaired in the posterior operation, and consequently there is a lessened tendency to hyperacidity, which is the important factor in the production of the ulcers. Jejunal ulcer manifests the same tendency to perforation as does duodenal ulcer, but the resulting peritonitis is more frequently circumscribed. According to Mayo Robson, general peritonitis is more likely to follow perforation after posterior gastrojejunostomy than after the anterior operation.

Uræmic Ulcers.—In 1859 Treitz described intestinal ulcers occurring in the course of nephritis which he attributed to irritation by the ammonium carbonate formed in the intestinal tract from the urea excreted into it. Since then the observation has been frequently confirmed, although some difference of opinion prevails as to the probable cause of the ulcerations. They have been variously attributed to capillary thromboses, to arteriosclerosis of the intestinal arteries, and to submucous hemorrhages. The latter view is the one held by Dickinson, but it has not met with general acceptance. In all probability the excretion of irritating substances into the intestinal tract is a factor more or less responsible for the ulceration. A feature lending considerable weight to this view is the fact that in the vast majority of cases of nephritis some degree of catarrhal enteritis is found, un-

¹ *Journal of Experimental Medicine*, vol. ii, p. 501

doubtedly caused by the irritation of excretory products; and various degrees of catarrhal and even diphtheritic enteritis are usually associated with the ulcerations. The ulcers are most frequently found and most numerous in the rectum, colon, and lower ileum, although occasionally they are almost or entirely limited to the duodenum. They vary greatly in size. The smaller ones have regular and somewhat undermined edges. They frequently coalesce, forming large, irregularly outlined areas of ulceration. Mathieu and Roux record a case of an ulcer thirty inches in length and involving the entire circumference of the bowel. The condition is usually met with in chronic interstitial rather than in the parenchymatous forms of nephritis, and according to Mathieu and Roux in subjects from eighteen to twenty-five years of age. The last statement does not represent the experience of the majority of observers.

Embolic and Thrombotic Ulcers.—The intestines are subject to changes dependent upon alterations of its blood supply, just as are other tissues of the body. The resulting lesions are dependent not only upon the character of the obstruction, but also upon its extent. If a large arterial branch be occluded so as to alter seriously the blood supply of a considerable part of the intestine, phenomena will result, the discussion of which will be considered in another place. If, however, smaller branches of the intestinal vessels be occluded, especially those running in the intestinal wall itself, ulceration will often ensue. Although thrombosis induced by sclerotic changes in the vessel walls may be the cause of such lesions, a much commoner cause is embolism, resulting from valvular diseases of the heart, abscess or thrombosis elsewhere in the body, or arteriosclerosis. Under those circumstances the character of the lesions in the intestinal wall will depend upon the character of the embolus, whether it be bland or infectious.

If an embolus occludes one of the smaller branches of an intestinal vessel, there soon develops a hemorrhagic infiltration of the submucous tissues which later extends to the mucosa. The area involved becomes swollen, firm, and dark or grayish red. On account of the interference with their nutrition, the tissues in the area of distribution of the vessel soon become necrotic, and the mucous membrane with more or less of the underlying tissues is cast off and an ulcer results. Usually the central portion of the area involved shows the most advanced changes. Frequently the visceral peritoneum corresponding to the area of involvement is the seat of a hemorrhagic infiltration. If the occlusion has been such that the entire thickness of the wall is involved, the ulcer becomes much deeper and early perforation may result. Usually the ulcers are of small size and involve only the mucous membrane and submucosa. They may be circular, girdle-shaped, or irregular in form. They are usually multiple. If the embolus be septic, the early changes may be the same as in the case of a bland embolus, but soon a localized submucous infiltration of leukocytes occurs and a small abscess forms. This increases in size and finally ruptures into the intestinal lumen. If the involvement of the wall be more extensive, perforation may occur and peritonitis result. Naturally whether the emboli be bland or infective, lesions in other organs similar in character to those in the intestines may be found.

Catarrhal Ulcers.—However inappropriate the term “catarrhal ulcers” may be, it expresses well enough the nature of the condition—an ulceration occurring in conjunction with and apparently the result of catarrhal inflam-

mation. It is seen especially in catarrhal enteritis of rather long duration, and more frequently in children than in adults. The ulcers are present in both the small and large intestines, but are more numerous in the latter. They are usually minute, round ulcers with very slightly indurated edges. Larger ones may occur which are frequently the result of the confluence of several small ulcers. These larger areas generally have an irregular outline. The ulceration usually involves only the mucous membrane, and at times not even its entire depth. The larger ulcers have a pigmented base, and at times somewhat indurated edges. The ulcers are usually numerous, and at times the process is so extensive that only islands of mucous membrane are left between the ulcerated areas. The mucous membrane that remains free of ulceration is the seat of catarrhal inflammation. Since the process is limited to the mucous layer, perforation practically never occurs; but it is not unusual to find various degrees of cicatrization when the condition shows a tendency to heal. Since the same cause is active in both conditions, follicular ulcers, to be described next, are at times found in association with catarrhal ulcers.

The mode of formation of catarrhal ulcers has been the subject of some discussion. The especially mooted question has been whether the primary stage of the process is in the mucosa itself, inducing ulceration by direct action upon the epithelium, or whether there is originally a submucous infiltration, inducing ulceration by interference with the nutrition of the overlying parts. Since the catarrhal changes preceding the ulceration are most marked in the mucous layer, and since only the most superficial portions of the mucosa may be ulcerated, the former theory is the more rational, and this view is supported by the histological studies of Woodward.

Follicular Ulceration.—Follicular ulceration occurs as a result of the same conditions that induce catarrhal ulceration. In the latter condition the mucous membrane is the portion especially affected, whereas in follicular ulceration the solitary lymphatic follicles are the seat of the process. The inflammation induces a hyperplasia of the lymphatic elements of the follicles, which later undergo central softening. The swelling of the lymphatic follicles interferes with the nutrition of the overlying mucosa and subjects it to mechanical irritation, so that it undergoes necrosis, and the softened elements of the lymphatic follicles escape, leaving an ulcer. As the extent of the follicle is usually greater than the area of superficial ulceration, the resulting ulcer has usually a more or less undermined edge. The ulcers are usually numerous, at times giving a typical honey-combed appearance to the part involved. If seen before ulceration has occurred the follicles appear on the mucosa as small, shot-like, yellowish prominences. Perforation seldom occurs. The colon and lower ileum are much more frequently the seat of the process than the upper portion of the intestinal tract, and the condition is more common in children than in adults.

Stercoral or Decubital Ulcers.—Stercoral or decubital ulcers are ulcerations resulting from the irritant action of inspissated faecal masses on the intestinal wall. This action may be purely mechanical, or, what is more probable, the mechanical injury to the mucosa serves only as the occasion for ingress of pathogenic organisms. The condition naturally is found only in the large intestines, and especially at the points where stagnation of the faecal current occurs, at the hepatic and splenic flexures, in the rectum, sigmoid, caecum, and appendix. The ulcers are usually circular in outline

and have an inflamed suppurating base. The condition is more frequently met in elderly individuals subject to constipation. Perforation of a stercoral ulcer has been known to occur. (See Diverticula.) Not infrequently these ulcers lead to cicatrization and stenosis, and Grawitz remarks that probably not infrequently these stenoses have been mistaken for carcinomatous strictures, while Nothnagel calls attention to the possibility of carcinoma developing as a secondary manifestation in the edges of the cicatrices, a process similar to the development of carcinoma following gastric ulcer.

Leukæmic Ulcers.—In leukæmia, and especially in the more acute form of the disease, it is possible for lymphatic hyperplasia in the intestinal wall to occur, of such a degree as to induce ulceration of the overlying mucous membrane. Such ulcers are more commonly found in the ileum than in other portions of the intestinal tract, although usually some other portions of the digestive tract, especially the mouth and pharynx, are involved. The ulcers are of irregular outline and have a ragged uneven base. Microscopically, this base is seen to consist of a dense mass of lymphocytes.

Trophic Ulcers.—In various nervous and mental disturbances, ulceration of the intestines has been observed. Nothnagel refers to cases reported by Kussmaul and Maier, Minkowski and Lorenz, in which extensive intestinal ulcerations occurred in conjunction with multiple neuritis. In most of these cases sclerotic or thrombotic changes in the vessels were found, so that Nothnagel concludes that the intestinal lesions were the result of the vascular changes associated with the neuritis. Hale White, Acland, and Targett consider it possible for the intestinal ulceration to result from disease of the brain and spinal cord, and quote cases to substantiate their claims. The cases of Targett and Ogle are particularly striking. In both of them there was fracture of the spine followed shortly after by diarrhœa, and at autopsy extensive ulceration of the colon was found. It is not improbable that these lesions were the result of the same factors inducing the superficial trophic ulcerations so common in spinal disease.

Ulcerative Colitis.—A number of the English clinicians, and especially Hale White, recognize a form of ulcerative colitis similar to but not identical with dysentery, occurring usually in asylums and hospitals, either epidemically or sporadically. The majority of observers claim the condition to be identical with dysentery, and their claim in regard to the epidemic cases is well supported by the work of Gemmel, Mott, and Vedder and Duval.

In regard to the sporadic cases, there is not as yet sufficient evidence to warrant the separation of the condition from dysentery.

Symptoms of Intestinal Ulceration.—In considering the symptoms of intestinal ulceration, duodenal ulcer, tuberculosis, and syphilis, as well as the ulceration of typhoid fever, dysentery and other specific infections are excluded.

There may be a complete absence of symptoms, even in cases of extensive ulceration, especially when the lesions mainly occupy the small intestine. When the colon is involved, and particularly when its lower portions or the sigmoid and rectum are affected, symptoms are less frequently wanting. Even in these cases, however, there may be a striking absence of clinical manifestations.

Among the symptoms met with in the different forms of ulceration of the bowels, diarrhœa, hemorrhage, or the passage of small amounts of blood,

the discharge of pus and shreds of tissue, anæmia, pain, fever, and general disturbances of health are the most important.

Diarrhœa is perhaps the most frequent of the symptoms. It is caused by the direct irritation of the intestinal nervous mechanism by the ulcerative process or by interference with intestinal absorption. Its character varies with the location of the ulcers. When these are situated in the small bowel, in the cæcum, or in the upper portions of the colon, diarrhœa, if present, is of the ordinary kind in which more or less fluid or unformed movements are passed. No special characters distinguish the diarrhœa in such cases from that encountered in simple irritations or catarrhal conditions. When the lower part of the large bowel is the seat of the ulcers, there is a greater tendency to admixture of mucus and blood, and the diarrhœa partakes of the nature of dysentery in proportion to the intensity and extent of the ulcerative processes. Intense straining at stool or tenesmus is sometimes met with in such cases, and marked soreness or tenderness over the descending colon or sigmoid flexure may occur.

Hemorrhage is less common in the various forms of ulceration now under consideration than in simple duodenal ulcer and typhoid ulceration. Sometimes, however, considerable hemorrhage or the passage of blood with each stool may occur, especially when the ulcers are situated in the lower bowel. Small amounts of blood and occult bleeding are more common. When sloughing attends extensive ulceration, blood, pus, and shreds of tissue may be passed together. Pus and portions of tissue are highly suggestive of ulcerative processes. They are more common in cases of dysentery than in other ulcers, but may occur in any form of ulceration that is extensive.

When intestinal ulceration is attended with large or repeated hemorrhages the patient becomes highly anæmic. In the earlier stages, and especially after large hemorrhages, the anæmia has the ordinary characters of posthemorrhagic secondary anæmia, but in cases of long-continued, slight bleeding a more intense anæmia results, and often one of a type that is highly suggestive of pernicious anæmia in the marked reduction of the number of erythrocytes, the altered character of the red cells, and the general appearance of the patient.

Pain is a symptom of no regularity of occurrence. Closely grouped ulcers in the sigmoid or rectum are commonly attended with marked local tenderness and pain of a constant or paroxysmal colicky type. Ulcers elsewhere in the bowel are less frequently attended with pain, and those in the small intestine rarely cause greater discomfort than that of occasional attacks of colic or vague soreness in the abdomen. When the ulcers are in the rectum or sigmoid, tenesmus may be pronounced.

Fever is a less pronounced symptom than might be expected. In stercoral and catarrhal ulcerations some degree of fever is usual, and at times it becomes marked. In the various other forms of ulceration it may occur, but is not regularly present.

The general condition of the patient is by no means indicative of the presence or degree of ulceration. Sometimes, despite extensive lesions, the patient presents no evidence of great disorder of health. In other cases, especially when diarrhœa, fever, and hemorrhage occur, there may be emaciation, loss of strength, and anæmia of a very significant grade.

Diagnosis.—It is extremely difficult to recognize intestinal ulceration in many cases. The occurrence of diarrhœa, the discharge of blood or pus and

blood, and local pain or tenderness are highly suggestive symptoms. When ulcers are situated in the rectum or sigmoid region they are readily demonstrable with the aid of the enteroscope. It may be doubted, however, whether the routine use of this instrument is advisable. Injuries to the ulcerated areas, even perforation of the bowel, have occurred to expert surgeons, and the cases in which a diagnosis can be made with certainty by the aid of the enteroscope are usually cases in which it can be arrived at with almost equal certainty without this instrument.

Prognosis.—Ordinary intestinal ulcers tend to heal in time, provided the proper hygienic, dietary, and medicinal measures are adopted. In cases in which extensive ulcers of the large bowel are present there is, however, a likelihood of great obstinacy to treatment. When extreme anæmia and emaciation have occurred the prognosis is rather unfavorable.

Treatment.—The treatment of ulcers in the upper part of the intestinal tract does not differ from that of diarrhœa due to other conditions. Careful regulation of diet, rest, local warmth and protection of the abdomen, mild astringents, such as bismuth salts, chalk mixture, vegetable preparations containing tannic acid, nitrate of silver, copper salts, or salts of lead, are the remedial measures to be employed.

Ulcers situated in the lower bowel can often be treated with advantage by injections or direct topical applications through an enteroscope. Among the useful forms of injection may be mentioned solutions of nitrate of silver (1 to 5000 to 1 to 1000), weak solutions of salicylic acid, boric acid, thymol, or other antiseptics, astringent lotions, such as mixtures containing hamamelis, hydrastis, kino, etc., or flushings with simple salt solution. Sometimes nightly injections of an ounce or several ounces of olive oil may be advantageous. In conjunction with such measures the regular use of mild laxatives such as salines, sulphur in combination with other laxatives, occasional courses of calomel or preparations of magnesia, have a useful purpose in preventing the formation of hard and irritating scybalous masses.

Direct topical treatment of the ulcers with stronger solutions of nitrate of silver and other astringents, suspensions of iodoform, and various antiseptics may prove effective in cases of limited ulceration so situated that it is readily reached through the enteroscope.

PERICOLITIS.

Occasionally inflammation occurs in the tissues surrounding the colon, resembling pericæcal and peri-appendicular inflammation.

Etiology.—The causes of this condition are numerous. Sometimes it results from direct injury; at other times, from organic lesions within the colon or perforation by foreign bodies. Probably in the majority of cases obstinate constipation and stercoral ulceration of the bowel are the causes of extension and involvement of the tissues surrounding the colon. Various foreign bodies, such as pieces of bone, needles or pins, fragments of straw or wood, and the like, have been discovered penetrating the wall of the colon or lying in an abscess that has formed outside. Attention is called, in the section on Diverticula, to the occurrence of inflammations within and surrounding such extensions from the bowel.

Pathology.—Whatever the original cause of the condition, the resulting lesions are similar, although varying in extent. Sometimes there is merely

an inflammation or ulceration within, and some reactive inflammatory thickening on the outer surface of the bowel. When more extensive destruction of the mucosa has occurred, or when complete penetration by a foreign body or perforation has taken place, more extensive external inflammation occurs. This may result in a chronic inflammatory thickening; or, if active infection has occurred, an abscess may follow. Sometimes, as Bland-Sutton has shown, such abscesses may discharge into the bowel, and the tumor, which was before palpable, spontaneously disappears. In many cases the final result of the condition is the formation of fibrous thickenings or of adhesions, which may attach the bowel to neighboring structures or cause contractions of various sorts.

The usual situations in which pericolitis occurs are the hepatic and splenic flexures of the colon or the sigmoid flexure. The term *pericolitis sinistra* has been especially applied to cases occurring in the last-named situation. Perityphlitis of cæcal origin is practically identical with the condition now under discussion; but, on account of its close relationship with appendicitis, it has been referred to in the discussion of that condition.

Symptoms.—The clinical manifestations indicate an acute inflammatory trouble in the affected region. Local pain and tenderness on pressure, more or less fever, and disturbance of the function of the bowel are the usual indications. In some cases constipation occurs as the result of inflammatory weakening of the affected segment of bowel or of pressure upon the bowel. In other cases diarrhœa occurs, and sometimes mucus and blood appear in the fæces. Sometimes a localized induration or mass may be discovered on palpation. In rare instances spontaneous disappearance of the mass may occur in consequence of rupture into the bowel.

Diagnosis.—The condition presents practically the same symptoms as those met with in cases of appendicitis. The location of the lesion usually excludes this condition; but the possibility of a misplaced, and even a left-sided, appendix should be remembered. Some cases in which a tumor has been felt through the abdominal wall have been regarded as cases of carcinoma; and the diagnosis has been disproved only after operation or the spontaneous recovery of the patient. The acute symptoms and the indications of inflammation and infection should, as a rule, aid in excluding carcinoma, when the possibility of tumors due to pericolitis is borne in mind.

Treatment.—The treatment of the condition is purely surgical.

APPENDICITIS.

Definition.—In a strict sense the term appendicitis should be used to define inflammations of the vermiform appendix. It has, however, obtained a broader clinical significance, embracing various inflammatory affections of the right iliac fossa with which inflammation of the appendix is associated as a more or less important factor. Formerly the term typhlitis was much in use, and was based upon the idea that inflammations of the region designated originated in the cæcum and head of the colon and spread from these structures to the surrounding tissues (perityphlitis). Modern investigations, surgical and pathological, have shown that in the great majority of instances perityphlitis has its origin in primary appendicitis, and for practical purposes it may be accepted as established that inflammations around the

head of the colon originate in the appendix, and should be dealt with as cases of appendicitis. The term appendicitis is sometimes loosely applied to diseased conditions of the appendix, such as cysts, mucocoeles, etc., which are not necessarily inflammatory, although secondary inflammatory conditions may subsequently associate themselves. When used in this way, the term must be recognized as having a clinical rather than a pathological applicability.

Notwithstanding the fact that various surgical writers have denied the existence of primary localized inflammations of the cæcum without appendicitis, there is abundant evidence that such a condition does occur. MacWilliams¹ reviews a number of cases reported in recent years, and shows conclusively the existence of both acute and chronic typhlitis independent of appendicitis, tuberculosis, dysentery, cancer, or other like conditions. These forms of typhlitis are due to obscure causes or to coprostasis. The disease of the cæcum may go on to ulceration, perforation, and the formation of perityphlitic abscess or general peritonitis, the appendix meanwhile remaining normal. H. A. Kelly himself has reported 14 such cases. For practical purposes, however, the comparative rarity of this condition justifies the physician in disregarding it, unless the development of the case has strongly suggested the retention of faecal matter in the cæcum and the physical signs indicate intestinal stasis in that region.

Anatomy of the Appendix.—Embryologically the appendix is a derivative of the caecal pouch, originating as a small process from that structure in the seventh or eighth week of foetal life. In the early stages of the development of the intestinal tube the cæcum is situated rather high up, near the under surface of the liver, but subsequently by elongation of the colon it assumes a lower position, carrying with it the developing appendix. Occasionally, the foetal situation of the cæcum and appendix is observed in later life. Usually in the adult the appendix lies in the right iliac fossa, its base or point of attachment to the cæcum being beneath the point described by McBurney and generally known by his name. This point is situated "exactly between an inch and a half and two inches from the anterior spinous process of the ileum in a straight line drawn from that process to the umbilicus." The attachment of the appendix to the cæcum varies considerably. In the majority of cases, or according to Treves in 90 per cent., a medioposterior position is discovered. Frequently, however, the point of attachment may be at the lower portion of the cæcum on the anterior or posterior surface, or on the outer or right surface.

To some extent the position assumed by the whole appendix is determined by its point of attachment. The position, according to Kelly and Hurdon in various cases, is as follows:

Horizontally, toward promontory or pointing laterally, 32 per cent.

Oblique, toward spleen, 10 per cent.

Ascending, 34 per cent.

Descending, 24 per cent.

The direction is determined not alone by the point of attachment, but also by the length and character of the meso-appendix, as well as by the size and character of the appendix itself. It is of clinical importance that the appendix not rarely occupies a position posterior to the cæcum and colon (in the retrocolic fossa). It is also important to recall that the appendix

¹ *Annals of Surgery*, June, 1907.

frequently extends down into the pelvis, and may be attached by its tip to pelvic structures.

Size.—The normal adult appendix varies in length from one-quarter of an inch to twelve and seven-eighths inches (Grauer). In the majority of cases the length is from one-half to three or four inches. The diameter, according to various authorities, ranges from one-eighth to one-third of an inch. Not rarely, however, these figures are exceeded, and sometimes appendices of extremely narrow caliber, perhaps mere thread-like formations, have been discovered. In these cases it is probable that secondary changes had reduced the size of the organ.

Shape.—Roughly speaking, the appendix may be described as a somewhat firm-walled, cylindrical tube, having a relatively equal diameter throughout its length from the caecal attachment to the free end or tip. The infantile or foetal type differs from this usual adult shape in presenting a funnel-like root of attachment to the caecum. The expanded, funnel-like base may terminate in a cylindrical appendix otherwise like that usually observed, but in some instances the whole appendix from its caecal attachment to its tip is roughly conical in form. Not infrequently constrictions or narrowings of the caliber of the appendix are observed. Generally these are the result of preëxisting disease and consequent cicatricial contraction, or the product of constricting bands of mesentery or of adhesions; but sometimes the contracted portions are of developmental origin.

Meso-appendix.—The mesentery of the appendix is a triangular or quadrilateral fold of the peritoneum arising from the under layer of the mesentery of the ileum and attached by its right border to the caecum and by its inferior margin to the appendix itself, usually extending about two-thirds the length of that organ from the caecum toward the free tip. The left border of the meso-appendix is free and semilunar or concave in outline. The shape of the mesentery varies to a great extent in different cases, and determines very largely the position of the appendix. Especially important in this direction is the length of its left or free border. The bloodvessels and nerves of the appendix traverse its mesentery, and there may be contained within this structure variable quantities of fat, as in other portions of the peritoneum and mesentery. Occasionally a lymphatic gland (Clado) is found near the root of the meso-appendix.

Considerable variation may be encountered in the shape and position of the meso-appendix, and these variations may determine certain peculiarities in the clinical manifestations of appendicitis. An extension of a serous fold from the appendix to the ovary (appendiculo-ovarian ligament of Clado) is of possible etiological importance. It is supposed to carry bloodvessels and lymphatics, which establish a communication between the circulations of the appendix and ovary. This vascular connection, however, has been denied by some experienced observers (Kelly and Hurdon).

Coats of the Appendix.—The coats of the appendix are four in number: (1) The peritoneal or serous; (2) the muscular, including an external longitudinal and an internal circular coat; (3) the submucous; and (4) the mucous.

The Serous Coat.—The peritoneal layer of the appendix is a rather firm membrane closely applied to the outer muscular coat excepting along the mesenteric attachment. Occasionally, also, on the side of the appendix opposite the attached mesentery, the serous coat is raised as a fold extending for some distance along the length of the organ. The superficial bloodvessels,

lymphatics, and nerves of the appendix lie in a looser subserous tissue. Normally the bloodvessels can be easily seen beneath the peritoneal coat, and when inflammatory congestion occurs the injection of the bloodvessels is very striking.

The longitudinal muscular coat of the appendix is a continuation of the muscular bands of the large intestine. This connection is of considerable practical importance, particularly as the anterior longitudinal band of the colon and cæcum is a direct guide to the root of the appendix. Contractions of the longitudinal coat may considerably shorten the appendix.

The circular muscular coat, somewhat less in diameter than the longitudinal, encircles the appendix, but according to Lockwood is deficient at the attachment of the meso-appendix (*hiatus*). This gap serves to transmit vessels, nerves, and lymphatics from the interior to the serous coat.

The submucous coat is of variable thickness from 0.2 to 0.8 mm., and is composed of loose connective tissue containing wide lymph spaces, lymphatics, and veins. It is separated from the mucous membrane by a thin, muscularis mucosa. The submucosa contains a considerable amount of elastic tissue, and the lymphadenoid tissue of the mucosa extends well into the submucosa in places.

The mucous coat is a redundant mucous membrane presenting on transverse section of the appendix a folded appearance and having a thickness of about 0.75 mm. On transverse section it is found to contain normal tubular glands or crypts of Lieberkühn and lymphatic follicles lying between the glands and extending from beneath the surface epithelium to and into the submucosa.

The surface epithelium of the mucous membrane is of columnar form, and many of the cells are of the goblet type. The same character of epithelium is found lining the crypts or glands and covering the lymphatic follicles.

When viewed on the surface with slight magnification, the openings of the crypts or glands are found arranged about the lymph follicles in a concentric fashion. It has been estimated that there are 25,000 glands in an average appendix (Kelly and Hurdon). The glands extend inward to the full depth of the mucosa, the bases often resting upon the submucosa. At their fundus there may be a bifid division and occasionally a division into three portions. The lymph follicles of the appendix are visible upon the surface of the mucous membrane as smooth spaces lying in the centre of the concentrically arranged openings of the crypts of Lieberkühn. The diameter of the follicles varies from 0.3 to 0.7 mm. They are most abundant in the depressions between the folds of the mucous membrane.

The lumen of the appendix is usually irregular, owing to the redundancy of the mucous membrane, but when accumulated contents cause distention of the organ in pathological conditions, the folds are obliterated by stretching and the lumen of the organ becomes circular.

Valve of Gerlach.—The opening of the appendix into the cæcum is somewhat like that of the ureter into the bladder; that is, an oblique one, with a fold of the mucous membrane on one side known as the valve of Gerlach. It has been supposed that this mucous fold or valve serves the purpose of preventing foreign bodies from entering the appendix—an opinion that, however, is by no means universally accepted or positively established.

Circulation.—The arterial supply of the appendix is derived mostly from the posterior ileocæcal artery. The branches supplying the appendix traverse

the meso-appendix, running toward the tip of the organ and distributing branches at various intervals. After reaching the appendix, according to Lockwood, they divide into external branches, which supply the serous coat and the muscular tunics, and internal branches, which, penetrating the muscular hiatuses, terminate in branches supplying the mucosa and sub-mucosa.

The veins accompany the arteries and terminate in the ileocolic and eventually discharge into the superior mesenteric vein. The return circulation, therefore, extends to the portal system. To a slight extent, however, some communication is established with the systemic circulation through the external colic veins.

The lymphatic circulation of the appendix is of great clinical importance. The capillaries begin under the epithelium of the mucosa and beneath the lymphatic follicles, and extending from the submucosa pass through the muscular coats to the peritoneal surface, where they terminate in large trunks accompanying the bloodvessels. They usually extend to the lymphatic glands lying posterior to the cæcum, and eventually pass onward to the mesenteric glands along the inner side of the colon. Reference has been made before to the small lymphatic gland described by Clado, situated in the base of the meso-appendix close to its attachment with the ileum and cæcum. In cases of appendicitis, lymphatic glands in other situations have been sometimes found enlarged, such as those along the external or common iliac vessels, and on this account other lymphatic connections have been assumed to exist. It is not improbable, however, that such enlargements of neighboring lymphatic glands may be the result of diffusion of soluble products of infection, independent of the regular lymphatic channels.

Nervous Supply.—The nerves of the appendix are derived from the superior mesenteric plexus and terminate in a plexus of Auerbach and a plexus of Meissner comparable to those met with in other portions of the intestinal tract. Through the solar plexus the nervous mechanism of the appendix is connected with the cerebrospinal system and with the right vagus.

Etiology.—Among the causes of appendicitis we may recognize those which are general and those which are determinative. The disease is distinctly one of early life, as is shown by all tables of statistics. The majority of cases occur from the eleventh to the thirtieth year of life inclusive. Between thirty-one and forty a fair proportion occur, while after forty years the disease rapidly diminishes in frequency, being rarely met with in the aged. It occurs in the first decennium of life in a small number of cases, and has even been recognized as a prenatal disease.

Sex plays a certain part, as the disease occurs in males two or three times more frequently than in females, according to all observers. Some speculation has been indulged in to explain the relatively greater frequency in males, but no satisfactory explanation can be said to have been reached. Some authors have believed that the additional blood supply obtained in females through the appendiculo-ovarian ligament prevents obliteration of the circulation, thus obviating an important cause of the disease.

Occupation is of no special importance, although it has sometimes been claimed that those whose occupation leads to irregularities in eating and to exposure are more liable to the disease than are others.

Nationality probably plays little part. A number of writers in this

country have pointed out the comparative rarity of the disease in the negro; but among other peoples the disease occurs with practically equal frequency.

Heredity is of no special consequence, although families have occasionally been described in which repeated cases have occurred. In this connection the possibility of simulation must be remembered.

Digestive disturbances are perhaps important in a general way, and, as will be shown below, have often a more direct bearing upon the occurrence of the disease. Doubtless chronic gastric disorders may occasion catarrhal conditions of the bowel, and may favor the development of appendicular infections. A more direct and local relation of gastro-intestinal diseases to involvement of the appendix will be shown to operate in cases in which the cæcum becomes distended or overfilled with contents.

Local Conditions.—Among the local causes, various anatomical conditions require consideration. It has been pointed out that the appendix normally opens by a relatively narrow mouth, guarded by a reflection of the mucous membrane of the cæcum known as the valve of Gerlach. In some instances the valve is wanting or the mouth of the appendix is larger than usual, so that the intestinal contents readily find access to the organ, and may be retained in consequence of narrowing lower down or of weakness in the musculature of the appendicular walls. In other instances the mouth, or opening, is contracted, and such contents as gain entrance to the lumen of the appendix in a fluid state may be retained after undergoing inspissation or solidification by the admixture of mucus, epithelial cells, and bacteria, so as to form hardened masses or concretions within the organ. In still other cases the arrangement of the meso-appendix or abnormal bands of adhesions that surround the organ at some part of its length, or stenoses and angulations in the organ, may similarly prove the cause of retention of material within the lumen. Sometimes such constricting conditions are so disposed that, while ordinarily of little effect in the direction of obstructing the discharge of the contents of the appendix, they may become wholly obstructive when the cæcum or other neighboring part of the intestine is distended, either with gas or with stagnating intestinal contents.

Whatever the cause of the obstruction, the result of retention of material is, according to some authorities, a rapid multiplication of the bacteria contained; and possibly, also, an increase in the virulence of the organisms concerned. This may occasion an infection of the mucous membrane and the subsequent changes that have been described as characteristic of the disease.

There can be little doubt that conditions that obstruct the lumen of the appendix at any part of its course are a frequent and potent cause of inflammations of the organ. Not rarely cases are met with in which the distal end, or half, of the appendix alone is involved, while the proximal portion, separated from the former by some obstructing band, angulation, or stenosis, is uninvolved. Cases are met with in which repeated attacks of moderate severity have occurred, with digestive disturbance and distention of the cæcum and colon, and in which the occasion of the appendicular involvement appears to be a temporary increase of angulation and retention of contents, without extensive infection or involvement of the mucosa and other coats. After a repetition of such attacks, a more severe involvement may occur; and the more completely obstructed distal portion may undergo rapid necrosis or gangrene.

Fæcal Concretions.—Formerly a great deal was said regarding the presence in the appendix of various foreign bodies, and also regarding the presence of faecal concretions. It is now generally recognized that such supposed foreign bodies as seeds, fruit stones, and the like are usually faecal concretions simulating such bodies. The significance of the occurrence of foreign bodies will be mentioned below. So far as faecal concretions are concerned it was formerly believed that these commonly enter the appendix from the cæcum, and then more or less immediately set up inflammatory conditions. The more modern view of the matter is that the concretions met with are usually produced within the appendix itself. They do not of necessity occasion any inflammatory change; although their presence is always a distinct menace, and their relation to the etiology of appendicitis is, in many cases, a very direct one. Concretions are formed by the inspissation of faecal matter retained in the appendix and by the admixture of mucus, desquamated and exudative cells, and multiplying bacteria. According to some investigators, the concretions are always the result of bacterial growth. They are mainly composed of faecal material, with certain mineral salts, such as phosphate and carbonate of lime and magnesia, and mucus. Not rarely they are stratified in much the same way as is commonly observed in gallstones. Such concretions are met with in a relatively large proportion of cases of appendicitis. Deaver found them in about 16 per cent. of his cases; Treves states that they occur in about 30 per cent. of cases; and in some other series the proportions have been even greater. According to Ribbert, they occurred in 38 of 400 appendices, or 9.5 per cent., taken without selection. As a rule, but one is found; occasionally there are several, and sometimes many have occurred.

Foreign Bodies.—Various foreign bodies, such as pins, small nails, portions of bone, seeds, and fruit pips, as well as other insoluble substances, may find their way into the appendix, and are occasionally met with in cases of appendicitis. Not rarely parasites have been found, such as *Trichiurus trichiura*, *Oxyuris vermicularis*, and *Ascaris lumbricoides*. Less commonly other and rare intestinal entozoa have been discovered.

Effects of Concretions and Foreign Bodies.—Although concretions may be met with in appendices that show but little, if any, evidence of inflammatory change, it must be considered that they are a potent cause of inflammatory conditions. In the first place, they are capable, through the direct pressure exerted upon the mucous membrane, of causing destructive changes in the epithelium, and thus opening the way for the invasion of microorganisms. This pressure effect is most likely to occur when a concretion has reached a considerable size and when, as the consequence of distention of the cæcum or other local conditions, an angulation is produced at the point of lodgement of the concretion. Again, the presence of a concretion may explain the retention of contents within the appendix, and thus may occasion multiplication of bacteria and infection of the mucosa of the obstructed organ. Frequently when gangrenous changes occur in the appendix it is found that the lesions have begun at the point of pressure from a concretion; and when perforation takes place, the opening is opposite the foreign body. In cases of peri-appendicular abscess, the concretions are not rarely found in the abscess cavity, having escaped from the appendix through such a perforation as has just been described.

With respect to foreign bodies of other sorts, it is undoubted that they are

capable of exercising irritative and more or less destructive effects upon the mucous membrane and thus of initiating an attack.

Influence of the Lymphoid Tissue.—The lymphoid tissue of the appendix is undoubtedly a factor of importance in the occurrence of the intense inflammations of this part of the intestinal tract. Not unlikely, the immunity of the aged to the disease is due to the involution that takes place in the lymphoid tissue, and its more or less complete disappearance in later life. No part of the gastro-intestinal tube is so abundantly supplied with lymphoid tissue except the tonsillar region, and it is noteworthy that these two situations are common seats of intense inflammation in young subjects. Sahli has introduced the phrase “angina of the vermiform appendix,” in recognition of this fact. It is not improbable that the rich supply of lymphoid tissue occasions a local susceptibility, so that irritating retained contents and multiplying bacteria more readily occasion intense inflammatory conditions here than elsewhere in the intestinal tract.

It is possible, also, that some as yet undiscovered function of the appendix, perhaps of a secretory nature, may play a part in the etiology. It has recently been shown that lesions of the colon may be caused by the subcutaneous injection into animals of soluble toxic bodies derived from cultures of dysentery bacilli, showing that the pathological changes in the disease itself may be the result of the excretion of the poisons generated by the organisms of the disease. In a similar manner, it may be that lesions of the appendix may result from the excretion through its mucous membrane of poisons generated by bacterial activity elsewhere in the gastro-intestinal tract.

The Bacteria of Appendicitis.—The microorganisms met with in cases of appendicitis are varied. By far the most frequent form is the *Bacillus coli communis*. All bacteriologists who have studied this question have recognized the preponderance of this organism. In many cases it has been found in pure culture; but usually some other organism is associated with it. Streptococci or staphylococci may occasionally be found without any associated germs. More commonly, however, they are found with other forms. The *Bacillus lactis aerogenes*, the *Bacillus capsulatus aerogenes*, the *Bacillus pyocyaneus*, the influenza bacillus, the bacillus of Friedländer, and a variety of others have been found either alone or in various combinations. It may be accepted as probable that in the majority of cases appendicitis represents a mixed infection, the most important organisms being the *Bacillus coli communis*, the *Streptococcus pyogenes*, and the anaërobic forms. Some observations make it seem probable that the milder cases with more definitely localized peritonitis and relatively benign clinical symptoms are principally due to colon infection, while the more intense cases are of streptococcic or anaërobic nature.

Pathology.—Various terms have been employed to designate what are sometimes described as different forms of appendicitis, such as simple, catarrhal, perforative, gangrenous, interstitial, obliterative. While these terms are applicable to different stages of the disease, they are undesirable if they convey an impression that they refer to different kinds of disease. In addition to this objection, it has been pointed out that unfortunate results have occurred from the impression that mild or moderate symptoms indicated a milder form of disease, such as would be suggested by the terms simple or catarrhal, whereas, in truth, the most serious pathological states of the organ may be present when symptoms have been practically wanting.

The beginning lesion of appendicitis may be assumed to be practically always in the mucosa itself, and in the great majority of all cases there is found in the appendix removed at operation merely a catarrhal or an ulcerative condition of the mucous membrane. In the least advanced cases slight changes are observed, such as shallow erosion with some accumulation of mucopurulent exudate and congestion of the small vessels in and beneath the mucous membrane. In the more serious grades extensive ulceration of the mucosa will be encountered. At the same time, involvement of the submucosa and of the other tunics may be met with. There may be very pronounced changes in the mucous membrane, with but little visible alteration of the exterior of the organ and but a little if any increase in its size. To this form or stage, with its almost exclusive involvement of the mucous membrane, the term catarrhal appendicitis is most applicable. Nearly always, however, when the mucosa has been considerably involved, extension to other coats will be found to have taken place. Microscopic examinations of the mucous membrane show in all stages and grades a tendency to necrosis of the lining epithelium with erosion or ulceration and infiltration of the deeper layers with polymorphonuclear leukocytes. At first the epithelial cells are separated and degenerate. Masses of bacteria may be seen penetrating between the cells and into the tubular glands. Later, considerable destruction of the epithelium occurs, and the glands themselves are distorted by the swelling and inflammatory infiltration and later destroyed by involvement of their own epithelium. Hyperplasia of the lymph follicles and infiltration with inflammatory cells are regularly present. The lumen of the appendix may become distended with accumulated secretions of a mucopurulent character, and sometimes the exudation becomes hemorrhagic from admixture of blood from the ulcerated surfaces. The accumulation of material within the lumen is favored by obstructions due to twists, kinks, contracting bands, stenosis, or foreign bodies within the appendix. The effect of these conditions upon the pathology as well as their role in the etiology will be referred to later.

In all but the mildest cases the submucosa is involved early in the disease; congestion of the vessels and, later, stasis of the blood and thrombosis with oedematous swelling constitute the most conspicuous lesions. In severe cases small or considerable hemorrhages in the submucosa are generally seen. The thrombosis of the vessels may occasion secondary hemorrhagic infarctions of the mucous membrane, and thus advance the whole pathological process.

The swelling and infiltration of the mucous and submucous coats soon occasion a hardening and rigidity of the muscular and serous coats in a direct mechanical way. This is particularly marked when obstructions near the mouth of the organ prevent the discharge of accumulating exudation, and thus occasion distention of the lumen. This hardening and distention of the appendix, to which the term erection has sometimes been applied, tends to increase existing kinks or other forms of obstruction, and thus leads to a more rapid involvement of the outer coats of the organ.

Whenever the mucous membrane and submucosa are considerably involved, it is customary to find at least slight disease of the serous membrane. In milder cases a little roughening of the usually smooth and glistening surface is observed, and is found to be due to inflammatory changes in the endothelial cells with beginning fibrinous exudation. The extension of the

inflammatory process from the interior of the appendix possibly occurs, as Lockwood has suggested, through the normal gaps in the muscular layer. It thus happens that a considerable degree of peritoneal involvement may occur before the muscular coat is to any great extent involved, except by distention and slight infiltration near the submucous junction. Eventually in many cases the muscularis becomes markedly infiltrated and degenerated, and necrotic changes cause a weakening by destruction of the muscle cells. Pressure of hardened faecal concretions or of foreign bodies within the appendix may determine the site of the earliest involvement of the muscular coats, and may be the occasion of a rupture at such a point. There is rarely extensive necrosis of the muscularis except when complete occlusion of the circulation through twists, bands, or other forms of obstruction has occurred.

Extensive purulent infection may occasion considerable involvement of the tunics or total gangrene of the organ. In other cases there may be considerable change in the mucosa and submucosa and considerable involvement of the serous coat, with relatively small grades of destructive change in the muscularis. Infiltration with leukocytes and other evidences of inflammatory change between the muscle cells is more usually encountered.

The *contents of the appendix* increase with more or less rapidity and in varying ways, according to the nature of the case. In some instances, with stricture or other complete obstruction of the outlet, an increasing purulent exudate may accumulate within the lumen and cause distention of the walls of the organ, eventually forming a pus-containing sac (empyema of the appendix). More usually the amount of exudation is less abundant, and there is a tendency to solidification of portions of retained faecal matter with exfoliated epithelium, mucus, and masses of bacteria forming small or large concretions. In many instances there are found within the appendix or within abscesses formed around this organ after perforation hardened faecal concretions evidently of older date. The part played by these in the etiology of the disease will be considered later.

The effect of increasing accumulation of contents in the lumen upon the walls of the organ is manifested in the obliteration of the folded plicæ of the mucous membrane and the conversion of the lumen from an H-shape to a more or less circular outline. With this stretching of the mucous membrane, there is a flattening of the various structures, follicles, glands, etc., and at the same time a similar effect is produced on the submucosa and outer tunics. In some instances the disease becomes quiescent after this stage has been reached, and subsequently an enlarged pus-containing cyst may be discovered when clinical symptoms have been practically wanting.

Obstruction to the lumen of the appendix antedating the attack or the result of inflammatory conditions of its walls has an important etiological relation. Similarly, congenital narrowing of the outlet, acquired changes in the mucous membrane of the cæcum involving the mouth of the appendix, or obstructions caused by strictures of the wall of the appendix due to previous attacks or by foreign bodies or faecal accumulations within the lumen play an important part. In all of these conditions retention of the contents may occur, the bacteria multiply and probably gain in virulence, and inflammatory conditions result. In other cases, the mouth of the appendix may be open and its lumen wide enough throughout its entire length to permit under ordinary circumstances of discharge of the contents or of any matter gaining entrance; but there may be at some point a slight narrowing due to a

stricture, a band of adhesion on the outside, a twist or a kink caused by a short mesentery, which may become increased by inflammatory swelling of the distal portion of the organ. As a consequence of this increased obstruction the contents and accumulating exudates are confined to the appendix, causing increased virulence of the proliferating organisms and increase of inflammation.

Extensive necrosis and gangrene of the appendix may occur very rapidly and sometimes with very slight involvement of surrounding parts. Complete occlusion of the organ at some one point, and especially by anatomical conditions or old adhesions, which exercise a constricting effect both on the organ and circulation, and unusual intensity of the microorganisms concerned in the etiology may be determining factors. Pressure of concretions or foreign bodies within the organ and thrombotic occlusion of the bloodvessels are other factors of importance. In these cases the mucous membrane is more or less extensively ulcerated. The contents of the appendix are composed of a fetid putrefying mass of mucus with exfoliated epithelium and other tissue elements and bacteria. The appendix presents the black or grayish and black appearance of necrosis and gangrene, and hemorrhagic extravasation under the serous coat and more or less fibrinous exudation upon the serosa are observed. The organ is friable and may tear upon the slightest manipulation. In many instances spontaneous rupture occurs and the contents are discharged into the peritoneal cavity or into a preformed abscess cavity surrounding the organ.

Involvement of Surrounding Structures.—The most important local result of appendicitis is peritonitis involving the folds and fossæ in the immediate vicinity of the appendix. In mild cases and when the infective cause is of a relatively low grade of virulence the serous coat of the appendix is covered with a slight deposit of fibrinous exudation. Such localized peritonitis does not of necessity indicate actual perforation of the organ with escape of infective material, but may occur from invasion of the organisms into the peritoneal coat through the muscular tunics. The peritoneal inflammation may extend to surrounding parts, and a more or less firm agglutination may take place. The process may advance no farther than this, and many cases of appendicitis after reaching this stage subside, leaving behind adhesions to adjacent structures. In more intensely infectious cases the amount of exudation is greater, and localized abscess cavities are formed between the appendix and the neighboring coils of intestine or the abdominal and pelvic walls. Minute perforations of the appendix after the formation of preliminary attachment by fibrinous exudate to adjacent parts hasten the formation of abscesses. On the other hand, a perforation of the organ without preliminary adhesion, and especially a sudden extensive perforation, may be followed by a widespread peritonitis with but little tendency to limitation in the form of a localized abscess. Doubtless the virulence and nature of the infective cause play a considerable part in the liability to rapid softening and necrosis of some part of the wall of the appendix, and at the same time determines the amount of fibrinous exudation that may be present. In the more virulent purulent infections, destructive effects upon the tissues are more marked and exudation of fibrin less pronounced.

Peri-appendicular Abscess.—The situation of abscesses resulting from appendicitis varies according to the position of the appendix and of the adjacent parts of the intestine. In the majority of cases the pus cavity is

found between the appendix and the ileum at its attachment to the cæcum, between the appendix and the head of the cæcum, behind the cæcum, or between the appendix and head of the cæcum and the right abdominal wall and iliac fossa. Less commonly it may be found in the pelvis, in the retrovesical fossa, or in Douglas' pouch. Extension of the infection to the upper part of the abdominal cavity is usually prevented by the omentum and small intestine. In some instances a long appendix lies posterior to the ascending colon and appendicitis may occasion an abscess at some part of its course behind the colon. The wall of the abscess is formed by the adherent appendix, adjacent intestines, other surrounding structures, and by more or less firm masses of fibrinous exudate. The contained pus is usually of ordinary consistence and frequently of fœtid odor from the action of organisms of the colon group and sometimes as a result of perforation of the appendix and actual extravasation of faecal matter.

The abscess may rupture in various directions. Most frequently it breaks through the abdominal wall. Next in order of frequency, according to A. O. J. Kelly, is perforation into the cæcum or other portions of the intestinal canal, into the general peritoneal cavity, into the pleural cavity, or into the bladder or uterus. In rare instances extension and perforation into the gall-bladder or ureter have been described. After such rupture inspissation of the remaining contents and of the walls of the abscess may occur and spontaneous cure result.

General peritonitis may result from the rupture of a primary abscess, or more immediately from perforation of the appendix in rapid gangrenous or necrotic types of infection. In some cases without perforation of the appendix virulent organisms may escape through the walls of the organ and occasion a general peritonitis, or after the formation of an abscess extension of the infection may occur without discoverable gross rupture of the abscess wall. In these cases of extensive and spreading generalized peritonitis the pus is apt to be thin and serofibrinous from the beginning, and there is but little formation of fibrinous exudate on the surface of the intestinal coils. Deep injection and a loss of the lustre of the peritoneal covering of the bowels is a characteristic appearance. In some, however, quite extensive coating with fibrin may be seen throughout the abdominal cavity.

Remote Lesions.—Aside from the customary lesions associated with any form of intense infection, such as myocarditis, infective nephritis, etc., various remote lesions may result from invasion of the infective organisms from the appendix into various surrounding structures. Septic thrombosis of the venules of the appendix is a common occurrence, but fortunately does not frequently extend beyond the smallest branches. In some cases, however, ascending thrombosis takes place and may occasion involvement of the larger mesenteric vessels and the portal vein. In these cases secondary pyæmic abscesses of the liver result. Extension may also take place through the retrocolic veins and sometimes through new-formed collateral branches of the systemic circulation, and may occasion secondary abscesses of the lungs.

Chronic Appendicitis.—After a severe attack of appendicitis, or more frequently after repeated attacks, changes may occur in the wall of the appendix and in the surrounding structures which have been described as chronic appendicitis. In a certain measure this term is justified, because the adhesions, distortions, and other conditions met with serve to maintain

a state of congestion and lasting catarrhal inflammation, of the mucous membrane, and perhaps also a slight chronic inflammation of the outer walls of the appendix. It would be more correct, however, to regard the conditions under consideration ordinarily as results of acute appendicitis rather than as a chronic inflammation in the strict sense. In cases of moderately severe non-perforative appendicitis, with formation of a limited amount of fibrinous exudate, adhesions probably frequently result after the acute inflammation has subsided. Even quite intense inflammations of the appendix and neighboring peritoneal folds may resolve, as is indicated by the discovery in many cases of dense fibrous adhesions that must have followed a previous intense attack of appendicitis with localized peritonitis.

Besides the adhesions which are found uniting the appendix to neighboring structures, and which incite to subsequent attacks of appendicitis, the organ itself is frequently found thickened, bent, or otherwise altered by the preceding acute inflammations. Appendicitis of this description furnishes most of the instances of so-called relapsing appendicitis, the thickened sclerotic walls in a measure protecting against perforation and the dangerous complications so commonly met with in more acute types.

Obliteration.—Obliteration of the appendix may be the result of previous inflammations, and is thus a form of chronic appendicitis. Some authors, however, have insisted that obliteration may be purely the result of involution in some instances. In cases of post-inflammatory obliteration, the lumen of the appendix is replaced by a cord of irregularly distributed cells and fibrous connective tissue surrounded by muscle and a serous outer coat. Sometimes areas of atypical disorganized mucous membrane are found, but in cases of complete occlusion all resemblance to the normal structure is lost.

Symptoms.—In a great majority of the cases of appendicitis the symptoms of onset and the clinical course of the disease are more or less similar and distinctive; so that, as a rule, but little doubt exists in the mind of the physician regarding the nature of the disease. As a result of the frequency of the disease, however, a small minority furnishes a sufficient number of instances of irregular onset and irregular clinical course to give the impression that the clinical features of appendicitis are quite as often puzzling as otherwise.

In typical instances, pain in the abdomen, diffuse at first, and limited to the right iliac region after a greater or lesser duration; rigidity of the abdominal walls, especially on the right side; nausea and perhaps active emesis; constipation; rigors, or definite chills, accompanied with or followed by fever, acceleration of the pulse, and a general feeling of profound illness, characterize the initial symptoms and the stage of fully developed appendicitis. Wide variations from this mode of onset and from the symptoms detailed are frequently encountered, and the symptomatology of the disease may be said to be as variable as that of any other inflammatory condition affecting the internal structures.

It would be impossible to distinguish groups of cases illustrating different modes of onset and different forms of evolution of the symptoms, and for this reason a better impression of the variable clinical course of the disease may be obtained by discussing the symptoms separately and referring to their variations.

Pain.—Probably the most frequent symptom is pain, which is rarely absent at some stage of the disease. It must not be forgotten, however,

that even in intense forms, such as those in which necrosis and gangrene occur, pain may be absent from the beginning to the end of the process. Nearly always the patient suffering with appendicitis is seized with a sudden, cramp-like abdominal pain, which at first occupies the central or umbilical portion of the abdomen, and subsequently moves toward the region of the right iliac fossa. There are cases in which, from the beginning, whatever pain is encountered is strictly localized in the right iliac region; and other cases occur in which the initial pain is vague and difficult to locate.

The nervous mechanism of the appendix explains to some extent the localization of the pain. It has been pointed out that the afferent nerves are connected with the solar plexus through the superior mesenteric, and that a connection is established with the splanchnic and the right vagus. The pain of beginning appendicitis is referred to the mid-abdominal region, on account of the distribution of the terminals of the splanchnic system to this area.

Not rarely, especially in cases in which the appendix has a position posterior to the cæcum, pain in the back and in a region posterior to the liver, and even as high as the right shoulder, may be encountered. When the appendix extends downward toward the pelvis, suprapubic, perineal, and ischiorectal pain may be complained of in the beginning, or after a preliminary period of general or mid-abdominal distress.

After the full development of the disease, pain and tenderness, as a rule, are in the right iliac region. There is often a lack of correspondence of the tenderness with the pain. The most extreme tenderness seems to occur in those cases in which the appendix lies relatively near to the anterior abdominal wall, and especially when it is bound by adhesions to the parietal peritoneum. In a recent case, in which the tip of the appendix was firmly attached to the anterior abdominal wall, and in which it could be felt as a small, rounded mass, so superficial that it was difficult to determine whether it was intra-abdominal or in the walls of the abdomen, the local tenderness was extreme. Similarly, in cases in which the appendix extends downward toward the pelvis and is attached to the bladder or the rectum, or to other pelvic structures, the degree of pain experienced during defecation, micturition, or digital rectal examination may be excessive.

Sometimes the pain is referred to distant parts, such as the thorax, the right thigh, the lumbar region, the back, or the upper portions of the abdomen. The position and attachments of the appendix may, to some extent, determine such referred pains, but this is not invariably the case.

In the fully developed stage, when pain, previously diffuse, has become localized in the iliac region, the area of involvement is usually limited, and, generally speaking, centres at McBurney's point. The character of the pain varies from a steady ache to a paroxysmal, cramp-like or colic-like condition. Tenderness in the same area is usually, but not necessarily, present.

Cutaneous hyperalgesia was first described as a manifestation of various visceral diseases by Head, and has been studied in connection with appendicitis by various physicians and surgeons. It appears to occur in nearly all primary attacks at some stage of the disease, except, perhaps, in the most violent acute forms. The cause of the sensory condition is probably the distention of the appendix. It has frequently been noted that the hyperalgesia suddenly subsides when perforation of the appendix has occurred.

In non-perforative cases the symptom gradually disappears. It is frequently present in second or subsequent attacks, but is often absent when the original attack has been sufficiently severe to destroy nervous tissue in the wall of the appendix. The area of hyperalgesia centres about McBurney's point, although it is not strictly confined to this region. More properly, it may be said to occupy the region of the eleventh dorsal area of Head, a band extending on the right side from the middle line below the umbilicus to the lumbar spine posteriorly. The hyperalgesia is determined by pinching or stroking the skin, beginning outside the suspected area and working toward it.

Tenderness must be distinguished from pain, although the two usually coincide. The degree of tenderness varies, as has been pointed out, with the character of the case and the location of the appendix. Its intensity varies from the mildest grades, in which it can scarcely be determined, to such severe degrees that the lightest touch or even the weight of the bed-clothes is intolerable. In these cases, as in all those in which painful sensations are prominent, the position occupied by the patient in bed is quite distinctive. He lies upon the back or lightly inclined to the right side, and flexes the right thigh upon the body so as to relieve to the greatest degree possible all tension upon the affected portion of the abdomen.

Intense pain, of a peculiar character, is found in those cases of acute appendicitis in which intestinal obstruction has occurred as a complication. The violent pathological peristalsis with tonic contraction of the bowel that occurs in this condition occasions intense paroxysms of pain, recurring from time to time with greater or less frequency. In the beginning, these pains may be strictly located near the seat of obstruction; but later they often become generalized, involving a considerable portion of the abdomen.

Rigidity.—Rigidity of the abdominal walls is a sign of extreme importance in the diagnosis of acute inflammatory lesions of the abdomen, whether affecting the appendix, the gall-bladder, or other structures near the abdominal walls. In appendicitis there may be a generalized rigidity in the early stages, when pain, also, is of a diffuse or generalized character. Later, when the sensory symptoms have localized themselves in the right iliac region, rigidity also becomes limited in its extent, and affects the muscles overlying the inflammatory area. A careful comparison of the abdominal walls on the right and left sides will—in most cases of appendicitis, at least—disclose a slight resistance, if not a definite rigidity, affecting the muscles over the diseased area. The degree of this depends upon the location of the appendix and the character of the surrounding lesions. When the organ lies anteriorly and in close relation with the abdominal wall, and when inflammation involves the structures between the appendix and the abdominal wall, the greatest degree of rigidity, as well as of tenderness, may be expected. There are, however, instances in which, notwithstanding an immediate relation of a diseased appendix with the abdominal wall, muscular rigidity is totally absent. When the appendix lies posterior to the cæcum, some spasm of the lumbar group of muscles on the right side may occur; but its detection is more difficult than is that of rigidity of the anterior abdominal muscles.

Reflex Symptoms.—Among the reflex symptoms, those affecting the stomach and the bowel are of greatest frequency and significance. Nearly always, in typical cases, nausea occurs. This may be attended with an

attack of vomiting at the very beginning of the disease, or some time after its onset; and repeated vomiting may take place. Not rarely, however, nausea occurs without vomiting, and may persist until the disease is well developed. Sometimes repeated vomiting may be a very conspicuous feature of the disease, and may overshadow pain, tenderness, rigidity, and other symptoms usually more distinctive in character. The character of the vomiting in the early stages of the disease is not peculiar. The contents of the stomach are ejected, with perhaps some bile. Intense, uncontrollable vomiting may terminate in stercoraceous discharges; but this is more usually the result of complicating conditions occurring in the later stages.

Constipation is usually a symptom of importance, although not invariably present. In the beginning its cause is found in the reflex nervous influences occasioned by intense pain. Later, inflammatory conditions in the right iliac fossa are mainly operative, by causing a local paresis of coils of intestine involved in the inflammatory area, coöperating with nervous inhibition due to pain. In the final stages, when diffuse or generalized peritonitis has occurred, constipation may be the result of a general paralytic condition of the bowels. Various local conditions connected with appendicitis may operate in a purely mechanical way. Thus adhesions between the appendix and the small or large bowel may produce obstruction; or large abscesses may compress and practically obliterate the lumen of the bowel. In some instances there is at first no disturbance in the action of the bowel, one or more normal movements occurring after the onset of the disease. In some cases, indeed, a diarrhoeal condition may exist for a time, but such cases, however, are rare, constipation being the rule.

Symptoms of Infection.—Symptoms of infection are usually met with in appendicitis, although there are cases in which, despite the existence of a definite infective lesion in this region, generalized symptoms fail to make their appearance. Rigors and chills have been mentioned as occurring in the onset of many cases. Sometimes these are pronounced, and are repeated from time to time after the beginning of the disease.

Fever usually appears in most cases, although it must be recognized as a symptom of uncertain value. In many cases of appendicitis, in spite of the seriousness of the local lesions, the temperature remains low, while in other conditions simulating this disease, fever is of at least as great frequency as in ordinary attacks of appendicitis. In the average cases the temperature soon rises after the onset, being as high as 100° to 102° F. within a few hours. Subsequently, a variable temperature is apt to mark the course. Often the temperature falls to the normal soon after the onset, without reference to the character of the local lesions. Sometimes a continued temperature may be present for several days, and may terminate in an irregular fever when peri-appendicular inflammation or abscess has developed. In most instances fever is but an uncertain indication; and the severest forms of appendicitis may be unaccompanied elevation of temperature from the beginning to the end.

Occasionally subnormal temperatures are encountered, and are usually significant of rather intense infections. On the other hand, extremely high temperatures may occur in severe infections with marked local reaction. Even extensive peritonitis may be unattended with elevated temperature.

In the later stages, when localized abscesses have formed, irregular movements of the temperature curve are usual, but by no means invariable.

A recent case in which with an abscess containing a large quantity of pus there was fever or leukocytosis may be quoted in this connection.

The *pulse* indicates the general condition of infection with somewhat more reliability than does the temperature. In many cases in which fever has been absent, rapidity of the pulse has, from the beginning, indicated the existence of a serious lesion. The character of the pulse varies with the nature of the infection and the local conditions. Acute inflammatory lesions with marked reaction are usually attended with a rapid, but relatively full and tense pulse. On the other hand, infections extending to the peritoneum, and especially diffuse peritoneal involvements, are accompanied by increasing rapidity of the pulse rate, and often with irregularity and loss of quality. Less intense peritoneal involvement sometimes affects the quality of the pulse in a different manner. In such cases a hard or wiry pulse of moderate rapidity and without any irregularity may be met with.

Increasing rapidity of the pulse, loss of quality, and the occurrence of irregularity are always grave indications in this disease.

Chills are sometimes met with at the beginning of appendicitis, or at various times throughout its course. A considerable proportion of cases begin with rigors or a sense of general coldness, after which nausea and the other symptoms set in. The chills usually cease after the disease has been fairly established, and there is no tendency to recurrence until extension of the infective process has caused invasion of the peritoneum. In cases of peri-appendicular abscess, regularly or irregularly recurrent chills may continue for a long time. Still more marked is the tendency to chills accompanying widespread infection, as in instances of pylephlebitis and pyæmic abscess of the liver.

Leukocytosis.—The condition of the blood is usually significant of an infective inflammatory lesion. In the great majority of the cases of appendicitis there is found a definite leukocytosis, the number of leukocytes usually exceeding 12,000 to 15,000 per cmm. The differential count shows a marked preponderance of the polymorphonuclear elements. Sometimes the proportion of these forms may reach extreme grades, such as 90 to 95 per cent. When localized peritonitis has developed, the leukocytosis may be increased to even greater figures; but a spreading peritonitis with intense general infection is apt to declare itself by a falling leukocyte count after a preliminary leukocytosis.

An increased number of the leukocytes is not, however, invariably present in appendicitis, any more than in infectious and inflammatory lesions elsewhere. Even in cases of abscess formation the leukocyte count may be normal or subnormal. Such cases undoubtedly are rare, although there are a considerable number of reported instances.

It has been maintained by some that a preponderance of polymorphonuclear elements without an actual numerical leukocytosis may occur in appendicitis, as in other infective lesions. Such a condition of affairs is at least rare, and its significance may be questioned.

Physical Signs.—Among the objective manifestations of appendicitis, those which attend the earliest stages are of the greatest importance, as prompt diagnosis is most essential to a proper conduct of the treatment. Rigidity has been mentioned and sufficiently discussed; it is undoubtedly a sign of extreme value. Somewhat later than this other objective evidences of the diseased condition of the appendix and the surrounding structures

may be found, among which tumor and abdominal distention are most significant.

Tumor.—At the time of the primary rigidity, a certain fulness in the right iliac fossa is usually detected. In part, this is the result of the rigid condition of the rectus muscle, which opposes a certain resistance to the palpating hand; and this gives the impression of a mass. If the tenderness is not too great, this rigidity may be overcome and the suggestion of a mass disappears. Very frequently, however, there will be found below the rectus muscle a rounded and somewhat yielding tumor-like resistance, caused by gaseous distention of the caput coli. The nature of this mass may usually be detected by percussion over it, when the tympany discloses the fact that the mass is an air-containing one. The percussion note, however, is not so purely tympanitic as that found on the opposite side, over the sigmoid flexure; probably on account of the thickening by congestion of the surrounding bowel walls and perhaps also the rigidity of the abdominal muscles.

In the later stages, when a peri-appendicular abscess has formed, this may be readily detected as a more or less resilient swelling, or sometimes as a firm mass, without any discoverable indication of fluctuation; but the character of the tumor differs mainly in consequence of its varying situation. When placed anteriorly to the appendix and cæcum, extreme tenderness to touch usually renders palpation more difficult and uncertain in its results; but, at the same time, more frequently enables the examiner to determine the existence of fluctuation, as well as the suggestive dusky discoloration of the skin and the subcutaneous cedema so frequently met with over abscesses in various parts of the body. Abscesses situated on the inner side, and posterior to the cæcum and appendix, cannot so readily be felt, usually cause less superficial tenderness, and are more likely to give the impression of considerable hardness. Not rarely it is difficult to distinguish such from solid growths by palpation alone. Abscesses extending downward toward the pelvis may be so deeply situated as to escape detection through the abdominal wall, but may be felt, upon vaginal or rectal examination, as resistant, elastic, or boggy swellings, to the right of the vaginal or rectal wall. The size of peri-appendicular abscesses varies greatly, the majority not exceeding the size of a lemon or an orange before rupture takes place. Sometimes, however, masses considerably greater than this are encountered, and may by their very magnitude prove deceptive. When external pointing takes place, the appearances presented are those usually met with in the case of the deep-seated collections of pus that have burrowed and reached the surface.

The Appendix Itself May Sometimes Be Palpable.—Some authors have claimed that this is frequently the case. Most writers, however, agree that a positive recognition of the enlarged or diseased appendix is seldom possible. In exceptional cases, however, it is exceedingly easy to feel some portion of the appendix or a large part of it. It is not difficult to be deceived, however, and to mistake inflammatory exudates, distended coils of intestine, etc., for the appendix itself. Occasionally, when the appendix is attached to the anterior abdominal wall, the attached portion may be felt as a mass that seems surprisingly near the surface. In other cases, the tip of the appendix has been felt high up on the right side of the rectum. More commonly masses here palpated are peri-appendiceal abscesses.

Abdominal Distention.—Distention of the intestines is usually an early symptom, and sometimes increases with great rapidity. Reference has been made to the fact that the caput coli is often slightly dilated in the beginning. At the same time, slight general meteorism may be observed. In later stages, increasing distention may take place from one of several causes. If the appendix is ruptured, or if an extension of the infection has occurred through the wall of the unruptured appendix, the result is peritonitis with more or less rapidly increasing tympany. The sequence of events here is that met with in all forms of acute peritonitis. In other cases the distention is the result of obstruction of the lower portion of the ileum, in consequence of violent local inflammation or of ileus resulting from inflammatory or other forms of obstruction. Rapidly increasing general distention occurs in these cases, as in other forms of acute obstruction of the bowel.

The absence of distention by no means excludes the existence of quite extensive local inflammation; and even when peritonitis has occurred, the abdomen may remain comparatively flat.

Vesical Symptoms.—Vesical symptoms are frequent. In the beginning there may be pain in the region of the bladder or retention of urine, as the result of nervous reflexes caused by the acuteness of the pain; and retention may persist throughout the whole course of acute cases. As a rule this disappears after the first day. Subsequently, and at times from the beginning, there is, on the contrary, a continuous desire for micturition, especially in those cases in which the appendix extends downward toward the pelvis, and in which pericystitis is present. In such instances the vesical symptoms may completely dominate the clinical picture.

Actual cystitis may develop as the result of a direct extension, or in consequence of general infection.

The urine, in cases of appendicitis, as in other forms of acute infective inflammation, frequently presents small quantities of albumin, with a moderate number of hyaline or granular casts. In some instances of greater severity a well-defined, infectious nephritis may occur, especially in cases of extensive abscess formation. Indicanuria is a common indication of the putrefactive fermentation that occurs as the result of the sluggish or constipated condition of the bowels and of the multiplying bacterial flora. Albumosuria has also been described as a symptom, resulting from the intestinal conditions or associated perityphlitic suppuration. It is a symptom of no regularity of occurrence and of no diagnostic value. In rare instances peri-appendicular abscesses have ruptured into the bladder or ureters, and large quantities of pus have been found in the urine.

General Peritonitis.—General peritonitis is the most ominous of the results of acute appendicitis. Unfortunately, this condition may develop very promptly in patients who have presented no symptoms of great gravity; and it is equally unfortunate that it may arise in the late stages after a somewhat benign course. In a small proportion the disease apparently begins with general peritonitis. The onset of the trouble is usually marked by somewhat abrupt indications, significant of rupture of the diseased appendix or of extension of infection to the peritoneum through the unruptured wall. Sharp pain or suddenly increased local tenderness and rigidity, rigors or chills, a rapidly increasing acceleration of the pulse, and elevation of the temperature, or, in cases of marked infection, a fall of temperature, with prostration and symptoms in general significant of shock, are mostly those which indicate

the beginning of this grave condition. Soon board-like rigidity of the abdominal muscles, intense tenderness or hyperæsthesia over the affected area, sometimes involving the whole abdominal wall, an anxious expression, a hard, small pulse, with increasing rapidity, and a dorsal decubitus, with a tendency to flexion of the right thigh, are significant of its further extension. Still later, the acute symptoms may ameliorate with the advancing infection and the overwhelming of the system with toxæmia. At the same time, increasing abdominal distention takes place; and the patient presents the facial and general appearance of one profoundly septic and having spreading abdominal disease.

Vomiting may mark the onset, and may continue throughout the course of the complication, until extreme prostration from septicæmia puts an end to this as well as to other acute symptoms. At first, the vomitus is not particularly distinctive; but very soon it becomes stercoraceous in character.

Septicæmia.—General septic infection may occur with or without extensive local lesions. As a rule, it is most marked in those cases in which the local infective lesion is decided. Irregular chills and high and irregular fever, with a rapid, weak pulse and a tendency to drenching sweats, indicate the existence of this complication.

Septic symptoms are most marked in those cases in which infective thrombosis extends from the diseased area through the mesenteric into the portal veins, establishing a suppurative pylephlebitis. In these cases, in addition to the evidences of a general septic process, pain is met with in the region of the liver; swelling of the liver may be detected, and jaundice appears. The last-named symptom may occur without pylephlebitis or pyæmic abscess, as the consequence of an intense septic infection, but is rarely marked in such instances.

Pyæmic abscess in other organs, such as the lungs, spleen, kidneys, acute swellings of the parotid gland, and acute infectious nephritis, are occasionally met with.

Subphrenic abscesses have been encountered in some cases, mainly as a result of extension of cellulitis behind the cæcum and colon. Pleurisy may follow the subphrenic condition, or may occur independently.

Clinical Varieties.—Attention was directed in the beginning of this discussion of appendicitis to the fact that the nature of the process is not essentially different in mild and severe cases, and that such terms as "simple appendicitis," "simple catarrhal appendicitis," "gangrenous appendicitis," and the like are in a certain sense mischievous if they create an impression that there are forms of appendicitis of essentially different characters, and that mild symptoms point to the existence of a benign process, while severe symptoms indicate a more serious disease. Practical experience has shown beyond question that the mildest of symptoms may be encountered in cases in which extensive necrosis or gangrene has occurred, while severe symptoms may be met with in cases in which a relatively slight disease of the appendix exists. However much the symptoms and pathological lesions fail to harmonize in certain cases, it cannot be denied that there are many instances of appendicitis in which the mucosa is involved to the practical exclusion of the other parts of the organ. Such cases are, properly speaking, catarrhal appendicitis. Doubtless many such cases occur without clinical symptoms of any sort, in proof of which the experience of the post-mortem table might be cited. A second group of cases are those in which

all the walls of the appendix are involved, with little or no extension outside; cases in which, in other words, the disease is confined to the appendix itself. A third group is that in which the disease has extended beyond the appendix and has involved the peritoneum or other surrounding structures, cases in which, in other words, peri-appendicitis, para-appendicitis, or perityphlitis has arisen. Finally there are cases in which a widespread general peritonitis is associated with appendicitis.

The infective agent may be the same in all of these forms, and the pathological changes do not differ in kind. The different clinical and pathological course is dependent upon varying local conditions affecting the circulation in the appendix and its drainage, and perhaps upon anatomical conditions promoting in some and preventing in other cases rapid extension of the infective process.

Appendicitis with Lesions Limited to the Interior of the Appendix.—There are cases of simple catarrhal appendicitis in which sudden abdominal pain, at first somewhat general, later limited to the right iliac fossa, slight tenderness and rigidity in the same regions, chilliness followed by slight fever and acceleration of the pulse, a little nausea and perhaps an attack of vomiting, and constipation make up the clinical picture. Sometimes, indeed, little beyond pain and tenderness occurs. After a variable period extending from a few hours to twenty-four or thirty-six hours the symptoms subside, leaving a variable degree of soreness that may persist for several days longer. The subsidence of symptoms is often coincident with natural or medicinal evacuation of the bowels. The experience of surgeons has shown that many such cases are instances of merely catarrhal inflammation, and the pathological lesions are often confined to the mucosa or mucosa and submucosa. The contents of the appendix consist of a scanty, viscid mucus mixed with some soft faecal matter and bacteria. At times, however, when some interference with the drainage of the appendix exists, as in cases presenting adhesions, kinks, angulations, or deformation in consequence of anatomical peculiarities, the contents may be more abundant and may be composed of somewhat inspissated faecal matter mixed with mucus and cellular exudates.

Formerly cases of this sort, in which sudden abdominal pain and tenderness are the principal symptoms, were described by some as cases of "appendicular colic," especially when fever and other manifest indications of inflammation were wanting. At the present time there seems little reason for believing that cases presenting the symptoms indicated are other than actual appendicitis, and even the possibility of a colic of this description arising in an uninfamed appendix must be doubted. Nor is it certain that the colicky pains of actual appendicitis are even due in large measure to peristaltic efforts on the part of the muscular layers of the appendix.

Unfortunately, the pathological conditions are not always as limited, as have been described, in cases presenting the symptoms indicated. There may be, with symptoms no more intense than these and with no greater duration so far as can be discovered, extensive necrosis or gangrene of the appendix and the presence of faecal concretions. Indeed, there is every reason to believe that appendicitis of the greatest gravity may be entirely latent, the first symptom appearing when perforation and peritonitis have occurred or peritonitis has supervened without perforation.

Appendicitis with Involvement of the Whole Thickness of the Organ.—In cases in which a rapid extension of infection causes almost immediate

involvement of the entire wall of the appendix the symptoms are usually more intense, the initial rigor or nausea more pronounced, the fever higher, the local symptoms more pronounced, and, in general, the patient's condition seems more serious. If subsidence of the symptoms occurs, this is more gradual and the local tenderness and rigidity are more prolonged.

In the simpler cases there is often a temporary amelioration of symptoms after free evacuation of the bowels, even though the purgation has no permanent good effect. Such relief is much less likely in the cases of extensive involvement of the walls than in cases of the catarrhal type. Associated involvement of the serous covering of the appendix adds to the gravity of the symptoms, and in a considerable proportion of cases is followed by extension of a localized peritonitis.

Appendicitis with Involvement of Surrounding Structures.—In some instances after an acute onset, moderate symptoms may continue for a period of from one to three or even five days, when an exacerbation sets in and evidences of extension of the infection present themselves. In these cases the local symptoms, as well as the fever, nausea, and vomiting, subside, and a few evacuations of the bowels may add to the apparent improvement. Gradually, however, or sometimes quite suddenly, a renewed chill or vomiting and the reappearance of sharp pain initiate the extension to the peri-appendicular structures. Cases presenting this course are probably in most instances caused by organisms of relatively low virulence, and occur in individuals whose general or local conditions offer considerable resistance to the infection. Unfortunately, however, the sequence of events is often far from being as satisfactory as this. In many instances there is a rapid extension with constantly increasing symptoms, and considerable localized peritonitis develops before any adequate amount of exudate can form to limit the spread of the infection by causing adhesions. After spreading to a certain distance, however, further extension may be prevented by exudation and agglutination of the bowels. In this manner general peritonitis may be prevented, and eventually a peri-appendicular abscess of considerable size may be formed. The symptoms significant of such extensions are usually quite marked, consisting of increasing local tenderness, rigidity, and swelling, increased but often irregular fever, general meteorism, with obstinate constipation, indications of cessation of local peristalsis, and finally increased vomiting, perhaps becoming stercoraceous in character.

In many cases the involvement of surrounding structures occurs suddenly after perforation of the wall of the appendix. The time of rupture may be indicated by an attack of renewed and severe pain, by a sudden feeling of sinking or prostration, nausea or vomiting, or fall of temperature and acceleration and arrhythmia of the pulse. In other cases no special symptoms occur, but the general features of the case take on an aspect of greater gravity and the local symptoms in particular become more marked. Soon the indications of a circumscribed or general peritonitis present themselves.

Chronic Appendicitis.—Two types of chronic appendicitis may be recognized. The first is one in which, as the result of a previous acute attack, general thickening of the walls of the appendix has remained, or a narrowing of the outlet, an angulation, or some other condition interfering with proper drainage. In these cases local or general abdominal symptoms may be more or less continuously present. In the second group, as a result of shortening or abnormal attachments of the meso-appendix, or in consequence of

angulations or adhesions, the appendix, although ordinarily free from inflammatory conditions, is prone, from time to time, to suffer when sluggishness of the bowels, gaseous distention of the cæcum, and especially faecal accumulations in the caput coli or cæcum occasion increased tension upon the appendix, increased angulation, etc. Under these abnormal conditions frequent mild, acute attacks may occur, and may be quickly relieved by free evacuation of the bowel or correction of the other conditions named. With each recurring attack, however, the appendix tends to become more distorted and damaged, and eventually a violent acute attack may terminate the condition with all the manifestations of a severe acute appendicitis. It is this form to which the term *relapsing appendicitis* has been particularly applied. While this name is used especially to designate cases in which acute appendicitis is supposed to occur from time to time in a relapsing manner, and without any special local condition persisting between the attacks, it is important to recognize that the type above described is probably equally common or more common; namely, that in which recurring attacks of increased tension or angulation take place without necessarily marked inflammatory conditions, until more severe lesions of the organ are finally established.

In chronic appendicitis of the first type, due to general thickening, adhesions, etc., persistent local soreness, increased from time to time when distention of the bowels occurs, a tendency to more or less troublesome constipation, sometimes an associated mucous or membranous colitis, and evidences of intestinal indigestion, with furred tongue, disturbed appetite, nausea, tympanitic distention, and vague generalized abdominal pains, are met with. Not infrequently, attacks of severe cramps or colic may occur, when the intestines are particularly sluggish.

In the relapsing type of chronic appendicitis there occur, from time to time, attacks indistinguishable from those of acute appendicitis, arising without any previous history of involvement of this region. In other cases there are, occasionally, short seizures in which intense localized pain and tenderness, with perhaps slight muscular rigidity, make their appearance without much fever, disturbance of the pulse, or constitutional disorder. When the attacks subside, the return to normal conditions is often so rapid that the thought of a severe local inflammation can hardly be entertained; and an examination of the blood in these cases usually reveals but little, if any, leukocytosis. It is frequently observed that the acute attack follows some very definite derangement of digestion; and the association of the latter, with the consequent localization of symptoms in the epigastrium or upper portions of the abdomen, and the presence of symptoms suggesting "biliousness," may readily lead to an overlooking of the appendicular disease.

The outcome of chronic appendicitis may be the development of a chronic intestinal disorder, such as membranous or mucous colitis or habitual constipation. In the cases of the recurrent type there may be a subsidence of the attacks after several have occurred, and even after attacks of intense violence. There is always, however, the danger that finally a very severe condition may develop; perforation or extension of inflammation to the peritoneum may occur; and the disease may terminate as do instances of violent primary appendicitis.

Diagnosis.—The symptoms of acute appendicitis, when marked, are so typical that a diagnosis can scarcely fail to be made. There are, how-

ever, many instances of obscure and even of latent appendicitis, in which no amount of clinical acumen or painstaking care will enable the practitioner to determine correctly the nature of the patient's disease, although suspicion may attach to the appendix. On the other hand, there is unfortunately a tendency that sometimes makes itself manifest to regard every form of abdominal pain as appendicitis, unless the evidence clearly points to some other disease. However common appendicitis is, and however important its early recognition, it would be a misfortune if practising physicians generally disregarded all that has been learned respecting the diagnosis of this disease by its symptoms, the physical signs, and the clinical examinations, to such an extent as to suspect its presence in all cases of abdominal pain, unless clear evidence of something else were at hand, merely because of the frequency and danger of the disease under consideration.

The development of sudden pain in the abdomen, followed by a localization in the right iliac region, and attended with rigidity of the muscles over this part, must be looked upon as having great significance. If, in addition, there has been a primary rigor, or nausea and vomiting, with constipation, the diagnosis becomes fairly assured. Fever is a symptom of little importance, and rapidity of the pulse, although a little more trustworthy as an indication, is not necessarily met with. When the pain becomes sharply localized in McBurney's point, and muscular rigidity is found in the same area, the diagnosis is practically established. The existence of leukocytosis is often an aid in differentiating between appendicitis and other painful affections in the same region, from which, indeed, many of the symptoms may be practically indistinguishable.

The Diagnosis of the Character and Stage of the Disease.—In discussing the varieties of appendicitis, attention has been called to the fact that it is often impossible to distinguish between mild appendicular lesions limited to the inner lining or to the walls of the appendix alone and cases of a severer grade, with beginning or even extensive peri-appendicitis. In many instances, however, a diagnosis of the grade of the disease is possible.

Simple catarrhal appendicitis is usually recognized by the mildness of the symptoms, by the tendency to rapid amelioration after evacuation of the bowel, and by the early return to normal condition. In cases, however, in which the symptoms are of equal moderateness, but tend to persist in spite of purgation, and in which even slight tenderness and fever persist, the probability of a more serious condition must be considered. Cases, too, in which the severity of the constitutional condition of the patient is distinctly out of proportion to the local manifestations may properly be suspected of being due to more intense, and perhaps necrotic or gangrenous, lesions of the appendix. Distinctly marked leukocytosis is an additional indication of severity and of probable extension of the infection to the peritoneal surface.

Peri-appendicular infection and the development of abscess may be indicated by the continuous increase of local symptoms and the appearance of tumefaction in the right iliac fossa; or by the recurrence of fever, pain, and rigidity after an interval of quiescence succeeding the initial attack. Sometimes the development of peri-appendicular abscess is so gradual and insidious, and the exudative material and agglutinated coils of intestine so thoroughly walled around the focus of disease, that an abscess of considerable size may develop with relatively no local symptoms; and there may be but little fever

and other constitutional reaction. In such instances, leukocytosis, also, may be wanting, even though an abscess of considerable dimensions has formed.

Extensions of the infective inflammation to neighboring veins or lymph channels, causing an ascending suppurative phlebitis of the mesenteric veins, and finally of the portal vein, or a lymphangitis of the retrocaecal region, are indicated by evidences of intense toxæmia and septicæmia and by localized symptoms of marked gravity. In the case of retrocaecal inflammations the pain may be confined to the back, and may extend upward toward the posterior aspect of the liver.

The development of a spreading peritonitis in appendicitis is indicated by intense constitutional disturbance. The general appearance of the patient is usually somewhat significant. Increasing local tenderness and swelling, with recurring rigors and elevation of the temperature, and with marked disturbance of the pulse rate, followed by general abdominal distention and a cessation of peristalsis, are among the important indications. Leukocytosis may at first rise to higher levels; but in many instances, especially those of severe infectiousness, the leukocyte count suffers an early fall. This may set in so promptly that, by the time the first examination of the blood can be made, a normal count, or even leukopenia, may be discovered.

Differential Diagnosis.—The onset of the appendicitis must be distinguished from that of a variety of abdominal conditions, any one of which may, in certain instances, bear so puzzling a resemblance as to make a certain diagnosis impossible.

Painful gastro-intestinal conditions of various sorts must, first of all, be distinguished. An attack of simple gastric indigestion or of acute gastritis frequently begins with symptoms like those of appendicitis. As a rule, the relation to some indiscretion of diet is more immediate and certain; the localization of the pain is more likely to be in the region of the stomach, and to remain in that place; nausea and vomiting and tenderness in the epigastrium are conspicuous; while fever and leukocytosis are less likely to be found. The latter symptoms, however, may occur, especially in children, and the local symptoms may be referred to lower parts of the abdomen, particularly when the gastric disturbance has, in some measure, been occasioned by a previous or coincident sluggish condition of the bowels.

Acute, painful affections of the small intestine more frequently bear a striking resemblance, and usually in proportion to the nearness of the approach of the irritative or inflammatory conditions to the lower part of the small intestine. Usually the pain and tenderness are more diffuse, and throughout the attack fail to become limited to the right iliac fossa. Rigidity is more general, when present; and the pains are of a more distinctly generalized colic-like character. Disturbance of the stomach is less marked, and diarrhœa more likely to occur. The condition is less liable to present the rapid development of grave symptoms met with in severe cases of appendicitis, and is less likely to undergo the early amelioration after free purgation seen in the mild cases of catarrhal appendicitis.

Intestinal colic, whether due to digestive disturbances or to inflammatory conditions of the intestines, may suggest an attack of appendicitis. (See Acute Enteritis.) The resemblance of lead colic is often very striking, on account of the association of obstinate constipation with the acute abdominal pain. The indications of localized inflammation, however, are wanting, and a

careful scrutiny of the history of the patient and a consideration of his occupation usually enable the physician to arrive at a satisfactory diagnosis.

Acute enteritis due to mineral poisoning, such as lead, mercury, or arsenic, as well as acute attacks of food poisoning, may simulate appendicitis, like other varieties of gastro-intestinal irritation or inflammation. They are distinguished by the general extensiveness of the abdominal symptoms, the absence of localized tenderness or rigidity, and the progressive prostration. Usually, also, there is more or less, and sometimes intense, diarrhœa.

Acute colitis resulting from stagnation of the contents of the large intestine and the accumulation of fecal masses in the cæcum, or following exposure to cold or digestive disturbances, may very well simulate an attack of appendicitis. This is particularly the case when the cæcum or the head of the colon is especially involved. In some instances an accurate diagnosis is impossible; and it is certain that in some of the cases the appendix becomes secondarily involved, when it must be obvious that the symptoms would bear even a more puzzling resemblance to those of independent appendicitis. Attacks of typhlitis due to stercoral accumulations usually present a less intense degree of local tenderness and rigidity, and are less acute in onset than appendicitis; but cases have been reported in which the local conditions have been violent, and in which stercoral ulcerations have caused perforation with the secondary development of perityphlitis, indistinguishable from that usually resulting from appendicitis.

Typhoid Fever.—The intestinal conditions in the early stages of typhoid fever may simulate appendicitis, especially in those instances in which the ulcers are closely grouped about the caput coli. Rigidity of the abdominal walls, marked local tenderness, and tumefaction occur in these cases; while fever and the general constitutional symptoms of the onset resemble those of many cases of appendicitis. The gradual onset after a week or more of indefinite malaise and gradually increasing fever, the complete anorexia, the dilated pupil, and the absence of leukocytosis, may aid in the proper recognition of the condition. Unfortunately, however, some instances of typhoid fever, with the particular localization of the lesions referred to, come on very abruptly, with marked symptoms closely resembling those of appendicitis. To add to the confusion, there is, in a certain number of cases of typhoid fever, an early involvement of the appendix, with the development of specific typhoid lesions in the lymphoid tissues of that structure. Such cases, of course, present peculiarly puzzling clinical manifestations.

In a certain number of cases of typhoid fever ordinary appendicitis may complicate the onset of the disease. It is not improbable that in these cases the specific lesions in the cæcum and the consequent interference with drainage of the appendix may have an important part in the etiology of the complicating appendicitis. The manifestations of the disease are similar to those met with independently of typhoid fever, but it may be said that, as a rule, these cases usually pursue a relatively benign course and become quiescent or are resolved. The histology and bacteriology of the appendix in these cases may show that the disease has not been due to specific typhoid lesions nor to invasion of the typhoid bacillus.

Acute intestinal obstruction, due to adhesions or fibrous bands, or to internal hernia, may in the beginning suggest appendicitis. The early development of indications of more or less complete intestinal obstruction.

with marked distention and without the usual evidences of inflammation found in appendicitis, will aid in the distinction.

Acute pancreatitis, the rupture of a gastric or duodenal ulcer, and acute peritonitis following rupture of ulcers of other parts of the intestinal tract suggest appendicitis in the suddenness of onset, the intense abdominal pain, the tendency to nausea and vomiting, the occurrence of fever, and in the constitutional condition of the patient. The localization of the symptoms and the profound character of the disturbance, especially the speedy development of the indications pointing to early collapse, enable one to arrive at a proper diagnosis, in the majority of cases, before the condition has proceeded to its more pronounced grades.

Sometimes, in the onset of acute infectious diseases, abdominal pain may suggest a diagnosis of appendicitis. Among these, pneumonia and malaria are important.

Pneumonia.—Not rarely, the pain in the early stages of pneumonia is referred to the abdomen, especially in the case of children, and a considerable number of cases have been observed in which the resemblance to appendicitis was so puzzling that operation was undertaken. A careful physical examination will, in most instances, prevent this error; but there are cases in which, owing to the lateness of the appearance of distinct physical signs, the diagnosis of pneumonia may be difficult; and the abdominal symptoms, therefore, may readily cause confusion.

Malaria.—Attention has been called by all systematic writers to the occasional onset of malaria with abdominal symptoms. In these cases the occurrence of a rigor and sudden fever, with intense abdominal pain, suggests appendicitis. There may even be rigidity and localized tenderness. In some instances the diagnosis has been possible only after an examination of the blood, suspicion having been created by the discovery of splenic enlargement or by the history of the case.

Diseases of the Gall-bladder.—Acute inflammations of the gall-bladder, biliary colic, and, less commonly, other conditions of the biliary passages are in many instances distinguished with difficulty from acute appendicitis.

Cholecystitis.—The pain of cholecystitis may be referred to the lower right quadrant of the abdomen, and whatever tenderness is discovered may be most decided in the same region. The symptoms of onset may be practically the same as those of acute appendicitis. On the other hand, in cases of appendicitis, the pain may be referred to the region of the gall-bladder; or, when the appendix lies posterior to the cæcum, it may be most marked in the back, posterior to the liver. Difficulty in diagnosis may, therefore, arise from the mistaking of cholecystitis for appendicitis, or the reverse. Usually the location of the pain and other symptoms and the discovery of tenderness and swelling in the region of the gall-bladder in the case of cholecystitis, or in the right iliac fossa in that of appendicitis, make a proper diagnosis possible.

When empyema of the gall-bladder has followed a cholecystitis, the tumor or mass may be found in a relatively low position, owing to the gradual increase in the size of the gall-bladder. Pain and tenderness are also situated in a lower position than is usual in the acute stages of cholecystitis. As a rule, however, the mass can be found to have a certain pyriform shape and to be dependent from the under surface of the liver; and the point of maximum pain or tenderness is usually above the transverse umbilical line.

Biliary colic simulates appendicitis in the suddenness of its onset, the intensity of the pain, and the associated rigidity of the abdominal muscles, together with nausea and other gastric symptoms. As in other hepatic and biliary conditions, however, the pain is situated higher up; and the development of jaundice is an important distinctive indication. Intense and repeated vomiting is never as frequent in biliary colic as in the average case of appendicitis.

Biliary cholelithiasis without definite attacks of biliary colic has often been mistaken for subacute or chronic appendicitis. The frequent occurrence of slight or severe pain in the right half of the abdomen, the pain not rarely being referred to the lower right quadrant, and the tendency to disturbance of the intestinal tract and to more or less gastric disorder are the suggestive symptoms. Not infrequently, in these cases, an enlargement of the gall-bladder, owing to its containing one or numerous gallstones and bile mixed with inflammatory exudate, is present. Suggestive tenderness in the region of the gall-bladder is one of the most important symptoms upon which a diagnosis may be made.

Nephrolithiasis and Renal Colic.—In acute attacks of renal colic, the intense pain radiating from the loin to the external genitalia and disturbances in micturition (temporary obstruction, followed by the appearance of blood-stained urine) are the symptoms of special importance in diagnosis.

When a calculus has gradually worked its way to the lower portion of the ureter, and has become fixed in this position it may occasion acute attacks from time to time, which are most difficult to distinguish from appendicitis. The pain and localized tenderness, if the right side is affected, may be in precisely the region of the appendix, and may show less tendency to radiation downward. The character of the pain, however, is often of a distinct cutting nature; disturbances of micturition (temporary stoppage, with swelling of the kidneys), the appearance of crystals or blood in the urine, and finally the results of *x-ray* examination, may establish the diagnosis.

Acute pyelitis and other inflammatory conditions of the kidney may be attended with considerable pain, and may, therefore, simulate appendicitis. The situation of the pain, however, the discovery of tenderness in the loin and perhaps enlargement of the kidney, and the results of an examination of the urine make the diagnosis possible in these instances.

Movable kidney has frequently been mistaken for appendicitis, the sudden occurrence of painful attacks (Dietl's crises) being the principal occasion of confusion. These attacks, in themselves, may be indistinguishable in their character or location from the cramp-like pains of a beginning appendicitis. A careful physical examination usually makes the diagnosis possible by discovering the more or less movable enlarged kidney. The greatest difficulty occurs in persons in whom the abdominal walls are sufficiently thick to prevent careful palpation, or in whom muscular rigidity is unusually pronounced. Among the distinguishing symptoms, the most important are those arising from disturbance in micturition. There may be a temporary elevation of temperature in Dietl's crises, but, as a rule, fever is absent.

Acute peritonitis from various causes other than those before mentioned may simulate appendicitis. Thus tuberculous peritonitis or peritonitis following perforations of the intestines or other hollow viscera of the abdomen, or consequent upon extension of pelvic infection, may resemble appendicitis superficially, and even on most careful examination. Pre-

ceding conditions, the location of the symptoms, the early evidence of more or less extensive involvement of the peritoneum, and the absence of the more or less localized involvement of the right iliac region, enable the physician to arrive at a correct diagnosis.

Salpingitis and *oöphoritis*, with or without localized peritonitis, may offer considerable difficulty in some instances. In these cases, however, the frequency of pain in the pelvic region and back, the occurrence of other pelvic symptoms, and the location of the acute tenderness are suggestive indications, while the results of vaginal examination usually make a correct diagnosis easy. The early development of a localized peritonitis in these cases, however, increases the difficulty of diagnosis, especially when the history of the earlier symptoms cannot be obtained or is uncertain.

Ovarian Neuralgia.—In some patients, particularly in neurotic young women, attacks of intense pain in the right lower quadrant of the abdomen may occur at the beginning of menstruation, and may persist through the whole period. Sometimes these seem to be dependent upon overloading of the cæcum, when a certain fulness will be detected on palpation. Moderate fever occasionally occurs. The resemblance to acute appendicitis may be very puzzling, especially when as a result of apprehensiveness, nervous mimicry of other symptoms, such as gastric disturbances and muscular rigidity, occur. A careful study of the patient's general condition, the absence of leukocytosis and of a characteristic clinical course may establish the diagnosis; but in some cases doubt must remain even after subsidence of the symptoms at the end of menstruation.

Nervous mimicry of appendicitis may also occur in women at other times and perhaps with nearly equal frequency in neurotic males. It must be remembered that there is a certain degree of physiological sensitiveness at the ileocæcal valve which may readily increase under nervous conditions, and abdominal rigidity is not infrequently met with under the same circumstances. The general indications of infection and local inflammation, however, are wanting, and if the nervous character of the patient is recognized a proper diagnosis is usually easy.

Prognosis.—Although the majority of patients with acute appendicitis recover, the disease must be regarded as one of decided seriousness, because it is quite often impossible to determine during the course of the disease that an unfavorable extension, which may terminate in rapidly fatal peritonitis, is about to take place; and because, in cases of recovery, such after-results remain that recurrent attacks will probably recur.

C Treated in a purely medical or tentative manner, the great majority of patients with appendicitis recover. It is impossible to quote figures that may be relied upon to express the exact truth, since it is extremely difficult to recognize milder cases with certainty, and sometimes even the severest forms would pass undiagnosed but for the character of the terminal conditions. The greatest difficulty is caused by the fact that there is a very large group of cases of extremely mild character in which pain, slight gastro-intestinal symptoms, possibly moderate fever, and rigidity in the side may occur, but in which the physician may hesitate to make a diagnosis of appendicitis. Practically all these recover, and very many of them certainly are never classified as instances of appendicitis. The result is that tables of prognosis fail to include at least a large number of instances of the disease, and consequently present a less favorable impression than

would be the case if all were included. Even with this error, which tends to make the outcome of the disease seem somewhat less favorable than it should be, the recoveries from appendicitis treated by ordinary medical means or by expectant methods constitute a very large proportion of the total number of the cases.

Sahli collected 7213 cases, of which 473 were operated on; while 6740 were not. Of the latter group, 91 per cent. recovered. Recurrences took place in 4593 cases; and of these, 3635 recovered without further recurrences. It must be recognized, in connection with such statistics, that it is probable that the cases in which operation was performed represented a disproportionately large percentage of the serious cases. On the other hand, cases in which the disease had advanced rapidly and operative measures seemed futile would be classified in the group of those not operated on.

Rotter has presented some very accurate statistics, based upon cases studied individually, to determine the features in each instance. Of 213 cases, 8.9 per cent. died. In 21 cases there was diffuse peritonitis at admission to the hospital. Of these, 7 or 33.3 per cent. recovered; while 14 or 66.6 per cent. died. The latter accounts for nearly two-thirds of the total mortality of 8.9 per cent. Of 192 cases with circumscribed perityphlitis, 156 (82 per cent.) recovered under medical treatment. Of the remaining 36 cases, 33 were operated on, and 2 died; 3 died without surgical intervention. There were, therefore, only 5 deaths (2.5 per cent.) among 192 cases of circumscribed perityphlitis (Nothnagel).

Treves states that the mortality of appendicitis, all cases being included, is not above 5 per cent. The outlook in cases in which perityphlitis has occurred is, of course, far less satisfactory, although the great majority of these also recover under expectant treatment.

A study of the surgical literature of the last few years convinces one of the fact that the mortality from acute appendicitis treated by prompt and skilful surgical methods is probably lower than that recorded in most of the statistics bearing upon the results obtained from medical or expectant methods. In a measure, these statistics may be somewhat deceptive, in that they fail to include some of the most serious cases, in which operation has been deemed inadvisable, and, on the other hand, include some cases of doubtful diagnosis. Objections of a similar nature might, however, be urged against statistics pointing the other way.

The liability to perityphlitis is always very great. Some instances occur of appendicitis without extension beyond the walls of the organ, but the tendency to extension through the walls or to perforation is extremely great. In those cases in which, as the result of an angulation, bend, adhesion, or other form of stenosis, temporary interference with drainage occurs when the cæcum is distended, mild catarrhal attacks may be met with from time to time, without very great liability to extension. Eventually, however, in these, a severe attack may occur, and may terminate in the usual manner.

The liability to diffuse peritonitis is decidedly less marked than that to perityphlitis. The mode of extension of the infection is such, and the reactive process in the neighboring peritoneum so prompt, that limitation of extension is usually brought about by the agglutination of neighboring coils of intestine and the formation of a restraining wall of more or less firmness. It is unfortunate that no method of diagnosis enables the physician to determine the existence of conditions that may lead to the

sudden rupture of an inflamed or a necrotic appendix, with the discharge of highly infective contents into the open peritoneal cavity and the consequent development of a sudden general peritonitis.

The liability to relapse after recovery from acute appendicitis is very great, although it cannot be accurately estimated in figures. There are many instances of recovery with no subsequent tendency to recurrence. This is especially true of those cases in which a total obliteration occurs. The usual manner of recovery, however, is by the formation of more or less fibrous thickening or adhesion, and such conditions predispose to subsequent reinfection.

Treatment.—It is difficult to state in a categorical manner whether the treatment of the disease should be invariably surgical, or only in selected instances. Medical men, as well as surgeons, recognizing that it is often impossible to determine when an apparently benign case will terminate in sudden extension or rupture, with the development of peritonitis, believe that it is usually wiser to regard every case of appendicitis as demanding surgical treatment. A few years ago it was urged by many surgeons that every case in which the diagnosis had been established demanded immediate operation, unless some unusual contra-indicating circumstance prohibited that method of treatment. This attitude was supported by statistical evidence that seemed to show that, taken all in all, more cases would recover if all were subjected to immediate operation than if an attempt were made to select those in which operation was desirable and to postpone operation in other cases. However much it may be urged, on the ground of such statistical evidence, that every case of appendicitis should be subjected to immediate operation, nearly all physicians and surgeons of experience do hesitate, in certain cases with mild and perhaps doubtful symptoms, and sometimes even in cases of frank although not threatening character, preferring, in such instances, to await the development of more definite evidences or of more imperative indications for operation, in the hope that, when the inflammation has become limited or has subsided, the safer interval operation may be performed. Recently, moreover, some surgeons of large experience, as Ochsner, advocate delay in operation in some cases of appendicitis with spreading infection. This view was formerly advocated mainly by European surgeons.

It happens, therefore, in practice, that among any series of cases a certain number will be regarded by both the physician and the surgeon as proper cases for non-operative treatment. In other instances the existence of some complication or coincident disease of a prohibitive character may prevent operation; and finally, certain patients positively refuse operation when it is proposed. Leaving out these exceptional cases the only safe plan in the treatment of acute appendicitis is immediate or prompt operation.

Medical Treatment.—The first appearance of symptoms suggestive of appendicitis demands immediate rest in bed and the utmost possible relaxation of the patient. In many cases the character of the symptoms of onset is such that the administration of a purgative may seem indicated before the symptoms have become marked enough to be more than suggestive. In view of the possibility of an acute appendicitis, the physician should in such instances select gentle measures, so as to avoid any unnecessary violence of peristaltic action. Enemata are more desirable than purgatives; and of the latter, a dose of castor oil, small doses of calomel, or the cautious

use of salines, administered at short intervals until effective, are preferable to a single large dose of the same remedies or to more active purgatives. After symptoms definitely indicative of appendicitis have appeared, the use of purgatives, even of the milder sort, requires careful consideration. The present view of most surgeons is opposed to their administration. Perhaps, however, such a generally sweeping rule is unwarranted. There are undoubtedly cases of mild type accompanying constipation and retention of feces or of gas in the cæcum and colon in which gentle purgation is both satisfactory and effective. Not rarely, in such instances, the free evacuation of the bowel may reëstablish adequate drainage and cut short an attack that might otherwise proceed to a more serious extent. Enemata may prove satisfactory, but with the tendency to intestinal sluggishness already existing, injections will often fail to relieve the higher portions of the large bowel.

Pain is one of the symptoms that usually requires especial consideration when medical treatment is demanded. In the beginning, when the nature of the pain is still somewhat doubtful, warm applications, such as large fomentations, hot-water bags, poultices, or stupes, may relieve pain, as well as gastric irritability. When the symptoms have become more localized in the region of the appendix, applications of cold are preferred; because of a possible controlling influence on the inflammation itself, and because they are rather more effective in controlling the pain. Ice-bags, cloths moistened with ice-cold water, ice poultices, etc., may be employed. They may be left in place continuously, or may be applied from time to time, for such periods as the tolerance of the patient will permit.

Formerly, leeches were frequently applied; and doubtless, in some instances, these give relief. They are rarely used at present, because, in case of a subsequent operation, the local wounds and effusion of blood produced add an undesirable complication at the site of operation.

The use of opium in the treatment of appendicitis has been generally discredited, because of its tendency to mask the symptoms and because in case of subsequent operation it so interferes with peristalsis as to complicate seriously postoperative conditions. Sometimes, however, despite theoretical and practical objections, the degree of pain is so great that small doses of opium are demanded when the diagnosis is still in doubt or when operation is contra-indicated. Certain surgeons advocate minimal doses, even in cases in which the diagnosis has been made and operation is contemplated. In no circumstances, however, should this drug be used in large amounts.

Occasionally, when gastric irritability, meteorism, and general intestinal colic are conspicuously associated with the other symptoms, carminatives, such as spirits of chloroform, Hoffmann's anodyne, menthol, oil of peppermint, phenol, etc., may give relief. Excessive gastric irritability may be controlled by small doses of cocaine, phenol, dilute hydrocyanic acid, cerium oxalate, or aromatic spirit of ammonium. In some cases in which operation cannot be performed, or in postoperative gastric distention and distress, lavage will occasionally put an end to troublesome or threatening symptoms.

Diet.—In the beginning stages, all food, and even water, should be withheld. If excessive thirst is complained of, small pieces of broken ice may be allowed; or, after evacuation of the lower bowel, enemata of warm water may be given to supply the needs of the system.

If acute symptoms have subsided after forty-eight hours, a cautious

return to liquid diet may be advisable. Milk or broth, in doses of from a dram to half an ounce, may first be given, and increased as the tolerance of the patient becomes apparent. A return of local symptoms, fever, or gastric disturbance should, of course, suggest the immediate withdrawal of food. This event should also suggest the desirability of immediate operative intervention, if operation had previously been merely postponed pending developments.

Treatment of Chronic Appendicitis.—Cases in which a history pointing to one or more previous attacks of appendicitis is obtained, and in which local symptoms and physical signs or disturbances of bowel function indicate the probability of adhesions, thickening, angulations, or other chronic alterations in and about the appendix, must be carefully considered with reference to the desirability of operation in the interval before the occurrence of another acute attack. When the diagnosis is certain, operation should be unhesitatingly recommended; and even if somewhat uncertain, operation is the safer plan when the occupation or life of the patient is liable to take him beyond the reach of immediate surgical aid should another acute attack occur. The risk from operation in the interval is exceedingly small, but the slight danger incident to general anaesthesia, the possibility of other accidents in the course of or following the operation, and the danger of postoperative hernia (not to mention the inconvenience, expense, and other undesirable features connected with an unnecessary operation) should prohibit exploratory incisions in cases with doubtful, even though somewhat suggestive, symptoms. It is better to await developments. Operations are sometimes recommended in cases in which obstinate abdominal symptoms of obscure character but not strongly indicative of appendicitis has occurred. Exploration will more likely fail than succeed in revealing any chronic disease of the appendix. In the great majority of cases, chronic conditions of the appendix furnish sufficiently clear indications to establish at least a presumptive diagnosis.

TUBERCULOSIS OF THE APPENDIX.

Tuberculosis of the appendix is occasionally met with at operations and at autopsy. It is said to be found in 2 per cent. of the operation cases (Lockwood). The condition is, like other forms of intestinal tuberculosis, usually secondary to tuberculosis of the lungs. It may occur independently of other involvement of the intestines, but is more commonly found in association with tuberculosis of the caecum. In a few instances, primary tuberculosis of the appendix has been met with. The lesions observed are of variable character. Usually there is a simple tuberculous ulceration; in other cases, extensive invasion of the walls of the appendix, with secondary caseation and pus-formation, may be found; while extensions to the peritoneum, in the form of miliary tubercles, or a more diffuse localized peritonitis, may be seen. Crowder has described a case of primary hyperplastic tuberculosis limited to the appendix, and similar in its appearance to hyperplastic tuberculosis of the caecum (q. v.).

Symptoms.—The symptoms of tuberculosis of the appendix may be indistinguishable from those of ordinary appendicitis; and a large number of the cases that have been reported have been discovered upon surgical operation for supposed appendicitis. In some cases a suspicion of the

nature of the process might arise from the fact that there is, at the time, decided enlargement of the regional lymphatic glands. A case of this sort has occurred in the experience of the writer, and the nature of it was readily recognized before operation.

In some cases secondary tuberculous abscesses form, and may burrow in various directions.

ACTINOMYCOSIS OF THE APPENDIX.

A number of instances of this condition have been reported. Spickenbaum has collected 27 cases from literature (Kelly and Hurdon). The symptoms are those of a sudden or gradually developing lesion in the right iliac fossa, with the development of persistent soreness and a tender swelling. Rarely, the process remains limited to the appendix. Usually it tends to spread to the neighboring structures.

CYSTIC DISTENTION AND MUCOCELE OF THE APPENDIX.

Retention cysts of the appendix are not uncommon. They result from the gradual transformation of the contents of the appendix, in cases of obstruction of its mouth or of obstruction of its lumen, into liquid of a serous or watery character. Considerable dilatation of the distal portion of the appendix may occur, and a cyst of sufficient size to be detected through the abdominal walls may be formed. The wall of the appendix suffers attenuation, and sometimes hernious protrusions of the mucous membrane between the separated fibers of muscularis may be seen. In most instances, such dropsical cysts of the appendix have been met with at operation or at autopsy, not having been suspected during life. In some cases, however, operations have been undertaken for suspected abdominal or pelvic tumors, and the conditions described have been found.

Mucocele of the Appendix.—Mucocele is a condition in which the appendix is found distended with grayish or whitish gelatinous material of a mucoid character. In some instances this may be the result of an inflammatory condition resulting from occlusion; in other cases, as those of Rokitansky, Draper, Vimont, Baillet, and Stengel, the histological features suggest a neoplastic process. In some of these cases the condition was found in association with papillomatous disease of the ovary.

TUMORS OF THE APPENDIX.

Various benign tumors have been found in or growing from the appendix, and among the malignant tumors, carcinoma and sarcoma.

Polyps.—Rarely polypoid outgrowths from the mucous membrane occur in the appendix as in other situations.

Myxoma.—One instance, shown by Churton and removed by Mayo-Robson, is referred to by Kelly and Hurdon.

Myoma.—A few instances are described in literature. Small nodular masses, about 5 mm. in diameter, occurred in the case described by A. O. J. Kelly. There was associated chronic inflammation of the appendix.

Malignant Tumors.—*Primary carcinoma* of the appendix occurs more frequently than is commonly believed. Kelly and Hurdon state that there are now on record 49 cases, including 2 designated as endothelioma. In most instances in which the situation was noted the growth occurred at or near the tip. The tumor is usually small, having been in 15 cases from 5 to 12 mm. in diameter. Larger masses have, however, been reported up to the size of a walnut. The gross appearance may be that of a fibrous tumor. Sometimes it has presented caseation, and has suggested tuberculosis. Microscopically, the appearances are those of a glandular intestinal carcinoma. Sometimes infiltration as far as the meso-appendix occurs, as in a case reported by Norris; 9 of the 11 cases studied by Kelly were of this description. Carcinoma of the appendix is somewhat peculiar in the early age at which it presents itself. Of 25 instances of primary carcinoma, 4 occurred under twenty years of age and 11 between twenty and thirty years. The youngest reported case is that of a girl aged twelve.

The clinical symptoms are practically indistinguishable from those of chronic appendicitis or of appendicitis with perforation. A fatal termination may be due to perforation and secondary peritonitis. Peri-appendicular abscesses are not uncommon. Extension of the carcinomatous process to surrounding structures occurs in a certain number of the cases.

The *prognosis* after removal has not, as yet, been certainly determined, because the time that has elapsed since the first operation for this condition was undertaken is relatively short. Halsted's patient, according to Kelly and Hurdon, was living five years after operation.

Secondary carcinoma of the appendix is occasionally met with, especially in association with carcinoma of the pelvic organs.

Sarcoma.—Sarcoma has been found in the cæcum and appendix in a few instances.

TUBERCULOSIS OF THE INTESTINES.

The role that the intestines play as a route of infection for the tubercle bacillus is discussed elsewhere, and requires no consideration here. It may be stated as an established fact that intestinal tuberculosis may result from the ingestion of tuberculous meat or milk. Under these conditions primary intestinal tuberculosis would result, as distinguished from secondary intestinal tuberculosis, which results from an infection of some part of the intestinal tract from a previously existing focus of tuberculosis. This is, in most cases, pulmonary tuberculosis, the intestinal lesion usually resulting from the swallowing of bacillus-bearing sputum. Less frequently, secondary tuberculous enteritis results from the extension of a tuberculous focus in the peritoneum, the abdominal lymph nodes, or one of the abdominal viscera.

Concerning the relative frequency of primary and secondary intestinal tuberculosis, Zahn¹ places the occurrence of primary intestinal tuberculosis at 2.27 per cent., while Ciechanowski,² on the basis of 13,203 autopsies, figures it at 1.04 per cent. Secondary intestinal tuberculosis Zahn found in 63.21 per cent. of all cases of pulmonary tuberculosis, whereas Grosser³

¹ *Münch. med. Wochenschrift*, 1902, Nr. 2, p. 49.

² *Wien. klin. Wochenschrift*, 1907, Nr. 37.

³ *Ein Fall von primärer Darmtuberkulose*, Inaug. Diss., Tübingen, 1900.

found it to exist in but 47 per cent. of the cases. In this connection the results of the investigations of Heller,¹ Councilman, Mallory and Pearce,² and Baginsky on the frequency of tuberculosis, especially of the digestive tract, in children dying of diphtheria, are interesting. Among 714 cases dying of diphtheria, Heller found tuberculosis in 140 (19.6 per cent.), in 53 (7.4 per cent.) of which it was primary in the digestive tract. Councilman, Mallory and Pearce in 220 cases found tuberculosis in 35 (16 per cent.), in 13 (5.9 per cent.) of which it was primary in the digestive tract. Among 806 diphtheria cases Baginsky³ found 144 (17 per cent.) to be tuberculous and in only 6 (0.7 per cent.) was it primary in the digestive tract. There is a convincing uniformity in these figures excepting for Baginsky's percentage of primary tuberculosis of the digestive tract. Heller cannot account for this discrepancy unless it be a result of the technique of inexperienced assistants.

Although the commonest lesion of intestinal tuberculosis is the tuberculous ulcer, other processes may present themselves, either in conjunction with ulceration or independent of it. Certain clinical distinctions may differentiate these cases and make it profitable to consider separately the ulcerative, the stenotic, and the hyperplastic varieties. Clinically, it may be found difficult to classify all cases strictly according to one or the other of these types, and frequently combinations of the types will exist together, but a proper understanding of their pathological characteristics is best accomplished by a separate consideration of the three varieties.

Tuberculous Ulcers.—The most frequent localization of tuberculous ulcers is in the ileum just above the ileocaecal valve. They occur, however, as high up as the duodenum and as low down as the rectum. The ulcer usually begins in a solitary follicle or Peyer's patch, although it may occur in the mucous membrane itself. It commences as a small, shot-like, gray nodule just below the mucous surface. This nodule enlarges and later its centre undergoes caseation. Observed microscopically, it will be seen to consist of a number of typical tubercles composed of giant cells, epithelioid and lymphoid cells, or of a diffuse caseating mass. Finally the entire nodule becomes caseous and subsequently breaks through the overlying mucous membrane, the caseous material is discharged into the bowel, and the tuberculous ulcer results. At this stage it has a small, crater-like opening, elevated caseous edges, and a caseous base. Frequently a number of these primary ulcers will unite to form a larger ulcer. In other cases the larger ulcers result from the simple increase in size of the single original ulcer, by the caseation of miliary tubercles deposited about its margins. Since this extension is by means of the lymph channels, the longest diameter of the ulcer is usually at right angles to the long axis of the intestine, and at times the ulcer extends as a girdle about the entire lumen of the bowel. The margins are elevated, irregular, and usually slightly undermined. Frequently, tubercles can be seen with the naked eye in the elevated thickened edges. The base appears as a yellowish, caseous mass, or as a grayish-red, granulating area containing numerous tubercles. The peritoneal coat of the ulcer is usually thickened, of a dark, bluish-gray color, and frequently

¹ *Deutsche med. Wochenschrift*, 1902, Nr. 39, p. 696.

² *Diphtheria*, Boston, 1901.

³ *Deutsche med. Wochenschrift*, 1902; *Vereinsbeilage*, Nr. 35, p. 270.

contains miliary tubercles, visible as minute, grayish-yellow, shot-like nodules. At times the peritoneum corresponding to the area of ulceration is covered by a fine exudation that may lead to adhesions between adjacent coils of intestines. The extent to which the ulceration may proceed varies greatly. Usually ulcers of varying size and age will be found in the same case. Now and then they become so extensive that small islands of healthy tissue are found surrounded by extensive areas of ulceration.

Terminal Changes.—Complete healing with the disappearance of all tubercles is an unusual occurrence. The ulcers frequently undergo partial organization, so that while some ulceration remains, a moderate degree of stenosis is also present. Perforation is seldom a complication of tuberculous ulceration, except in the rectum. Two factors tend toward this infrequency: the thickening of the peritoneal covering and the tendency to the formation of peritoneal adhesions. It is consequently especially in young, rapidly progressive ulcers that the tendency to perforation is greatest. If perforation occurs, either a general peritonitis results, or, if adhesions between adjacent coils of intestines have formed, walling off the seat of perforation, a localized peritoneal abscess results. On account of the fact that the ulcers are most frequent about the ileocecal valve and usually most advanced in this area, the site of the perforation is relatively most frequently found in the right iliac fossa, or in the pelvis where the coils of the lower ileum usually lie. Occasionally multiple perforations have been found. Perforation has been known to occur into neighboring organs as the bladder or uterus. Ulcers in the rectum not rarely perforate and cause ischio-rectal abscesses. Carcinoma has been known to develop in the site of an old tuberculous ulcer of the intestines.

Symptoms and Diagnosis.—The symptoms of ulcerative tuberculous enteritis do not differ materially from those of simple enteritis or of other forms of ulceration of the bowels. The most constant and characteristic symptom is diarrhoea, but this is by no means invariably present even when there is quite extensive ulceration. The stools are either soft and unformed, although presenting no special alteration from the normal in other respects, or thin and watery. Mucus may be found in small masses, or as strings or shreds, especially in the cases in which the faeces are semi-solid. There may be admixture of blood in small quantities, and occasionally considerable hemorrhage occurs. When formed stools are passed, or when the faeces are hard from constipation, they may be coated with blood-streaked mucus and pus, provided the ulceration is situated in the lower bowel. Continued diarrhoea causes rapid emaciation and loss of strength, although occasionally the patient maintains his flesh and strength surprisingly. Soreness and localized tenderness are not usually marked unless extension to the peritoneum has occurred. In that event indications of more or less pronounced peritonitis may supervene. Ordinarily the patient's temperature is not much influenced by tuberculous enteritis. The fever met with is usually due to the primary pulmonary lesion. Sometimes it becomes more marked when intestinal ulceration has occurred.

Since the symptoms of tuberculous ulceration of the intestines do not in themselves differ materially from those of most of the chronic inflammatory and ulcerative conditions in the intestines, the diagnosis must rest to a great extent on the associated phenomena.

Due attention must be paid to the fact that in children primary intestinal tuberculosis is much more common than in adults, and that more than 50 per cent. of the cases of pulmonary tuberculosis in adults are complicated by intestinal ulceration. Consequently the occurrence of diarrhœa and other symptoms of enteritis in an adult suffering from pulmonary tuberculosis would justify strong suspicions of the existence of intestinal ulceration. At the same time it must be recalled that amyloid disease of the intestines, the most prominent symptom of which is diarrhœa, is a common complication of tuberculosis. The diarrhœa of amyloid disease is usually more watery than that of tuberculous enteritis, and is less commonly associated with the presence of occult or visible blood.

In children the diagnosis is usually more difficult than in adults. A persistent diarrhœa, with progressive wasting, abdominal pain and distention, and enlarged glands without determinable cause, would warrant a tentative diagnosis of tuberculous enteritis. In both adults and children the diagnosis would be greatly strengthened by the discovery of tubercle bacilli in the stools or a positive tuberculin reaction. It must be remembered, however, that when infected sputum is swallowed tubercle bacilli may possibly be found in the fæces before an intestinal lesion has occurred.

Stenosing Tuberculous Enteritis.—Stenosis of the intestines of tuberculous origin without ulceration results when the tendency to organization as a result of the inflammation exceeds the tendency to destruction of tissue. It is by no means a frequent condition, but probably not so uncommon as has been formerly considered. A distinction must be made between those cases in which stricture has succeeded upon ulceration and those in which the stenosis is the primary and predominating feature. The latter constitute the type now specially under discussion. The tuberculous nature of these strictures is sometimes determined only with the greatest difficulty. Fibiger reports two such cases which were shown to be tuberculous only after exhaustive microscopic examination. He thinks that in the past many of these cases have been looked upon as syphilitic. The strictures are most frequently found in the ileum and colon and are usually multiple. In a case reported by Grosser both the ascending and transverse portions of the colon were involved, while in Hartmann's case the cæcum was intensely stenosed. Erdheim reported four cases of multiple tuberculous strictures of the ileum. The symptoms are those of chronic intestinal obstruction, and the tuberculous nature of the condition can be diagnosed only by the associated lesions or by exclusion of other etiological factors. The tuberculin or ophthalmotuberculin reaction may naturally be of great assistance in the diagnosis.

Chronic Hyperplastic Intestinal Tuberculosis.—This is a form of tuberculosis of the intestines that has attracted considerable attention during recent years. According to Conrath, it was first recognized by Durant in 1890. The condition is characterized pathologically by varying degrees of stenosis and ulceration, but primarily by a proliferation of tuberculous granulation tissue in the intestinal wall. This leads to a great increase in thickness of the wall and consequent stenosis, which produces one of the predominating clinical manifestations. The condition may originate, according to Hemmeter, in either the mucosa or the serosa, and is usually secondary to tuberculosis elsewhere. Its almost invariable site is the ileocæcal region.

The predominance of the hyperplastic over the destructive processes is probably a result of decreased virulence of the tubercle bacillus.

According to Conrath, the majority of the cases occur between the ages of twenty and forty years. The active symptoms are preceded by a long duration of vague, rather mild intestinal disturbances. Not until the stenosis is sufficient to produce symptoms of obstruction is the condition possible of diagnosis. These obstructive symptoms are of insidious onset and form one of the most constant features that the condition presents. A tumor presents itself sooner or later, and the resemblance to carcinoma is often marked. Obrastzow looks upon the slow and late development of the stenosis as pointing to tuberculosis rather than to carcinoma. The mass usually has a cylindrical form, giving the impression of a greatly thickened intestinal wall. Occult blood is usually present in the stools, and frequently the faeces contain visible quantities of admixed blood. Krokiewicz claims that the diazo reaction is much more frequently present in ileocaecal hyperplastic tuberculosis than in carcinoma. Fever is usually an accompaniment of the process. Naturally the diagnosis is greatly substantiated by the presence of tuberculosis elsewhere in the body, by a positive reaction to tuberculin, or by the discovery of tubercle bacilli in the stools.

Treatment of Intestinal Tuberculosis.—The prevention of intestinal infection is to some extent possible. Patients suffering from pulmonary tuberculosis should be made to realize the danger of swallowing sputum. Unfortunately, this occurs during sleep, and cannot then be prevented. The main purpose of treatment after ulceration has occurred should be to save the patient's strength by controlling diarrhoea and as far as possible the ulceration itself. There is some tendency toward healing of the ulcers, but the object of treatment is not so much to secure this result as it is to control the waste of the patient's vitality. Careful regulation of the diet is an essential. This may necessitate some diminution in the amount of food that might otherwise be desirable, but careful consideration is necessary to decide whether it is wisest to limit feeding on account of the bowels or to continue overnutrition on account of the pulmonary and general infection. No set rules can be laid down. Pasteurization or predigestion of the milk is often desirable. Irritating vegetables should be prohibited and broths, meat, and highly seasoned foods should be used with caution or prohibited.

So far as remedies are concerned, it is always well to use those that are least likely to disturb the stomach. Small doses of bismuth subnitrate, subgallate, or salicylate given rather frequently and continued for some time may have a useful effect. Among the antiseptics, guaiacol carbonate, creosote, carbolic acid, ichthyol, or iodoform may be used. When diarrhoea is excessive, small doses of nitrate of silver, acetate of lead, and vegetable astringents may be administered temporarily or until the bowels become more settled. Sulphur seems to exercise a useful effect in some cases.

When ulcers are found in the rectum, direct treatment through a speculum or enteroscope may aid healing and prevent extension to the perirectal tissues.

The stenosing and hyperplastic forms of tuberculous enteritis demand surgical treatment if the symptoms are pronounced and the condition of the patient permits of operation. The results in some cases have been very satisfactory.

SYPHILIS OF THE INTESTINES.

Intestinal lesions of syphilis are rare. They occur in both congenital and acquired syphilis but are much more common in the former.

The intestinal lesions of congenital syphilis are always associated with syphilitic phenomena in other organs. They usually take the form of multiple small gummata, and are found almost solely in the small intestines, and especially in the ileum. They take the form of flat, grayish elevations, at times involving the solitary follicles or Peyer's patches, at times not. Superficial necrosis frequently occurs, leaving an ulcer with a fibrous base and very slightly elevated edges. The ulcers usually extend in a direction at right angles to the axis of the bowel, and at times lead to stenosis. Kundrat and Moerck have reported the perforation of a syphilitic ulcer.

In acquired syphilis, symptoms of enteritis at times occur early in the disease which may be of the nature of intestinal manifestations comparable to the secondary cutaneous manifestations. Pathological observations of the condition are wanting.

Later in the disease intestinal lesions are observed similar to those observed in congenital syphilis, excepting that they are more common in the large intestine than in the small. They appear, first, as grayish-red, smooth, gummatus elevations, which later undergo necrosis resulting in flat, sharply marginated ulcers, with a smooth, translucent, gray or yellow base. As is the case with the congenital lesions, they tend to extend around the lumen of the bowel and cicatrize, thus producing stenosis. Occasionally perforation occurs.

The most frequent situation of syphilitic ulceration of the intestines is in the rectum and particularly in its terminal portion just above the sphincter. The ulcers are distinguished from those of dysentery by their smooth, gray base and the tendency to induration of the edges and extensive stenosis. It is probable that stenosing, tuberculous ulcers of the rectum have been frequently mistaken for syphilitic lesions. Syphilitic rectal stenoses are more common in women than in men, and according to Bäumlér are most frequent between the ages of seventeen and thirty years. They may result from the cicatrization of a primary lesion, of mucous patches, or of ulcers that have been caused by necrotic gummata.

The *symptoms* of syphilitic involvement of the intestines are those of simple intestinal ulceration or stricture and permit a probable diagnosis only on the basis of the history or the associated findings of syphilis, and possibly their response to the therapeutic test. Syphilitic stenoses of the rectum are distinguished by the marked induration and extensive cicatrization.

The *treatment* of syphilis of the intestines is directed to the general infection and to the control of the diarrhœa. The latter often persists in spite of ordinary treatment and until active antisypilitic remedies are employed.

AMYLOID DISEASE OF THE INTESTINES.

Amyloid disease of the intestines occurs as a result of the same causes as produce amyloidosis elsewhere. The most prominent of these causes are chronic tuberculosis, syphilis, chronic suppuration, and the various cachexias.

In point of frequency, the intestines stand fourth among the organs affected, the kidneys, spleen, and liver being the organs more frequently involved. Usually the entire intestinal tract is diffusely affected, but at times the ileum only is the seat of the disease. More rarely the colon alone is affected. The process may involve any or all of the tissues composing the intestinal wall, but seldom affects the mucous membrane itself. The lymphoid follicles usually remain entirely free from the amyloid involvement. The diseased intestine presents a pale, shiny, translucent appearance, and on application of iodine gives the typical amyloid reaction by turning brownish red and when subsequently treated by sulphuric acid, becoming blue or violet.

A feature of amyloid disease of the intestines requiring special mention is the so-called *amyloid ulcer*. These are described as ulcers varying in size from that of a pinpoint to that of large areas involving the entire circumference of the intestines in a girdle-like fashion, and from 5 to 15 cm. in length. They have smooth, slightly thickened edges and a pale base with numerous small striations. Colberg considers them to be of frequent occurrence in conjunction with amyloidosis of the intestines. He explains them as originating from the breaking off of the brittle villi by the mechanical effect of the intestinal contents. The majority of observers do not agree with Colberg as to the frequency of amyloid ulcers. It is possible that a simple ulceration occurs as a result of the insufficient nutrition of the surface epithelium caused by the amyloid degeneration of the underlying vessels. Moreover, it is not improbable that some of the ulcers described as amyloid were tuberculous ulcerations occurring as one of the manifestations of the disease producing the amyloidosis.

Mild degrees of amyloid disease of the intestines present no *symptoms* by which it can be recognized clinically. More severe grades of the process present one constant feature—diarrhœa. The stools are moderately frequent and watery, but present no characteristic features by which the condition can be positively diagnosed. The cause of this watery diarrhœa has been variously attributed to anæmia of the intestinal wall, to increased permeability of the vessel walls, and to decreased absorptive powers of the intestinal mucosa. The question of the presence of blood in the stools in amyloidosis of the intestines is one of considerable importance. Colberg, claiming amyloid ulcers to be a common association of diffuse amyloid disease of the intestines, looks upon the presence of blood in the stools as a diagnostic feature of the condition. The probabilities are, however, that the presence of blood in the stools points to the existence of other conditions than simple amyloidosis. Pain and tenderness play no part in the symptomatology of uncomplicated amyloid disease of the intestines. The *treatment* of the condition resolves itself into the treatment of the primary disease and an attempt to control the diarrhœa, which is best accomplished by the mineral and vegetable astringents and opium.

SPRUE.

Sprue, or psilosis, is a form of chronic inflammation of the gastro-intestinal tract met with in certain tropical countries—notably in the Orient—characterized by great weakness and wasting, anæmia, and a chronic form of diarrhœa.

Etiology.—The essential nature of the disease has not yet been determined. It was at one time thought to be due to the *Anguillula stercoralis*, but this view has been abandoned. According to Scheube, it never occurs epidemically and is not contagious. The disease occurs only in hot climates and affects Europeans who have resided for some time in such climates more frequently than natives. It is equally common in the two sexes, differing in this respect from hill diarrhoea, which is more frequent in men than in women.

The disease may occur without any predisposing cause, but often follows exhausting conditions, such as affections of the digestive tract like dysentery and other diarrhoeas. It is said to occur most frequently among women in those who have suffered with uterine troubles, especially such as have been attended with hemorrhage. Van de Scheer believes that there is some connection between disease of the appendix and the occurrence of sprue. Cantlie suggests that it may have some relation to scurvy, because fresh meat, fruit, and milk exercise a curative influence in some cases.

The geographical distribution is very wide. It is common in Southern China and Southeastern Asia generally, but has been met with in Africa, Ceylon, and the West Indies. Occasional cases appearing in England, Europe, and America may be traced to a previous residence in the tropics.

Pathology.—The lesions of sprue consist in a catarrhal inflammation, followed by erosions and atrophic changes affecting the whole gastro-intestinal tract. The mucous membrane of the mouth may present an inflamed surface, with erosions and aphthous spots. The small intestine is usually extensively involved, and the colon may present distinct ulcerations. When it has continued for some time, secondary changes may be met with, such as marked atrophy of the mucous membranes, areas of distinct erosion, and sometimes small, cystic dilatations of the intestinal glands. Enlargement of the mesenteric lymph nodes is frequent. Secondary changes in the pancreas have been described; and Manson believes that there is a functional sluggishness of the liver.

Symptoms.—The onset may be gradual or abrupt. In many cases it occurs in the course of other diarrhoeal diseases. The patient presents a wasted, anæmic appearance, and on examination of the blood the evidences of severe secondary anæmia are found. Abdominal distention occurs, and may cause a reduction in the extent of liver dulness.

The most characteristic features of the disease are those affecting the gastro-intestinal tract. The tongue and mouth become sore from exfoliation of the mucous membrane and the formation of herpetic or aphthous lesions. The term "Ceylon sore mouth" has been applied to this condition. Dyspeptic symptoms and flatulent distention of the stomach are frequent. The characteristic diarrhoea soon develops, and continues, with exacerbations and periods of quiescence, throughout the disease. The stools are large, usually loose, of a light grayish or white color, and frequently frothy. Chemical examinations show an increase of mucus and some albumin, as well as an excessive quantity of fat. Quantitative analyses indicate that intestinal absorption is much diminished. Notwithstanding the light color, a urobilin reaction may be obtained; and the color of the faeces is probably largely due to the excess of fat, although Manson has suggested functional inactivity of the liver as perhaps a contributing cause.

The clinical course of the disease is a chronic and remitting one, periods of active intestinal symptoms alternating with intermissions of quiescence.

Prognosis.—Cases recognized and treated early usually recover, although the tendency to relapses is very pronounced. Fatal cases terminate as the result of continued diarrhœa, lack of digestion and absorption of food, and consequent inanition.

Treatment.—Strict regulation of the diet offers the best chance of recovery, particularly if instituted early. A strict milk diet kept up for several weeks appears to be most generally advisable. Cantlie has recommended a pure meat diet, and sometimes the administration of fresh fruit or berries. Drugs, excepting such as are used for symptomatic treatment, have little value.

HILL DIARRHŒA.

Hill diarrhœa is a form of disease frequently met with among Europeans who have lived in India and have been transferred to a hill station. Changes of temperature, or of pressure, and other conditions incident to the change of station are regarded as the causes that determine the occurrence of the disease. The specific cause, if any, has not been discovered. The disease is more common in men than in women.

The *symptoms* of this disease consist in the passage of large, loose, colorless stools, generally more abundant in the early morning hours, and associated with dyspeptic symptoms and flatulence. The disease usually ends in a few days or weeks, but may require a return to lower altitudes before it subsides.

EPIDEMIC GANGRENOUS PROCTITIS.

This disease, also known by the names *El Bicho* and *Caribi*, or the Indian sickness, is characterized by a spreading phagedæna of the rectum or colon. It has been met with in Guiana, Venezuela, and Brazil, and probably occurs in other countries. It developed among the Indians of Guiana in a serious epidemic form in 1856, but Clemow doubts whether it exists there at the present time. In some cases an epidemic gangrenous stomatitis has been associated with it.

INTESTINAL OBSTRUCTION.

Definition.—The term intestinal obstruction is here used to signify incomplete or complete interference with the onward movement of the intestinal contents and their eventual discharge, occasioned by some form of mechanical or structural impediment of more or less limited extent. The term does not apply to interference with the passage of the intestinal contents caused by the general weakness or paralysis of the bowels or by the conditions of the contents themselves, which are referred to in the discussion of *Constipation* and *Intestinal Obstruction due to Motor Paralysis of the Bowel*.

A variety of terms have been introduced to designate different kinds of obstruction, and are, therefore, in a limited sense synonymous with obstruction.

Ileus is an ancient name, variously applied to paralytic conditions of the intestines, to obstruction in a general sense, or to obstruction with feculent vomiting. It is rarely used at the present day, and on account of its indefiniteness should be abandoned.

Occlusion implies a complete closure of the intestinal lumen.

Stricture signifies a contraction of the lumen caused by disease of the wall of the gut, usually involving its entire circumference in a more or less limited section.

Constriction signifies a narrowing by adhesions, fibrous bands, or similar conditions outside the bowel.

Obturation signifies an obstruction of the lumen of the intestines by contents, foreign bodies, or other conditions inside the bowel.

Compression indicates obstruction due to the pressure of tumors, misplaced organs, or other conditions outside the bowel.

Incarceration indicates an obstruction, more or less complete, from the retention of a coil of intestine beneath fibrous bands, within hernial sacs, etc.

Strangulation indicates the constriction of a coil of intestine in which more or less complete obstruction of the bloodvessels supplying this coil has taken place, causing intense venous stasis.

Intestinal obstruction may be acute or chronic. Frequently, after partial chronic obstruction, there is an acute increase of the obstruction, and clinically, difficulty is experienced in distinguishing such cases from purely acute obstructions.

General Pathological Anatomy.—Certain conditions affecting the bowel and its immediate connections, the peritoneum, the mesentery, etc., result from obstructions of all sorts without reference to the precise morbid condition that has occasioned the obstruction. The special pathological anatomy of obstruction will be considered in connection with the individual forms of the disease.

Characteristic differences are met with in the anatomical conditions occurring in chronic cases, as contrasted with those of acute character.

Chronic Obstruction.—A striking contrast is seen in the condition of the bowel below and above the point of obstruction. Below the obstruction the intestinal coils are empty and contracted, and their color more or less grayish or white. The condition is that seen in the empty intestine of persons who have died of inanition. Sometimes remnants of the intestinal contents remain in the contracted bowel below the obstruction, especially when hardened fecal masses had been present at the time of the obstruction and, on account of their physical characters, could not readily be transported onward. Above the obstruction, the bowel is distended, and, after a certain length of time, elongation, thickening from hypertrophy of the walls, ulceration, and other inflammatory changes in the mucous membrane and in the serous coat may be met with.

The first effect of obstruction is the accumulation of contents above the constricted area, and a resulting dilatation of the bowel. Later, this distention seeks higher and higher levels, until almost the entire intestinal tract above the constriction may be involved. The distended bowel contains fluid or somewhat consolidated fecal matter, according to the situation of the obstruction. In cases involving the small intestine, fluid contents alone are met with. In obstruction of the large intestine, there may be

semisolid or even solid faecal masses. This, however, is not invariable, as the contents may be fluid, even in obstruction of the lower part of the large bowel. A certain amount of gas may be present, but distinct accumulations of gas are unusual, as the gases are absorbed from the mucous membrane so long as the circulation of the bowel has not been seriously disturbed. Moreover, gases are usually able to pass beyond the obstructed area after any onward movement of fluid or solid faeces has become impossible.

Hypertrophy of the wall of the bowel follows as the result of the obstruction and distention and the increased peristaltic efforts of the bowel to overcome the condition. This hypertrophy affects the muscularis especially, and may be confined to this part, although some thickening of the other tunics may also occur. Experiments in animals have shown that the hypertrophy begins as early as the fourth or fifth day after obstruction of the bowel has been established, and is quite marked by the ninth day (Herczel). The hypertrophy in question is due to an increase in the width of the muscle fibers, rather than to increase in their number. It is a functional hypertrophy, which, to a certain extent, enables the bowel to overcome the obstruction, and in some of the chronic cases it undoubtedly serves to maintain a partially adequate movement of the faeces for some time.

It has been maintained by some that the hypertrophy is the result of ulceration of the mucous membrane rather than of the obstruction itself. This view, however, will scarcely meet with general approbation.

Changes in the mucous membrane result from the stasis of the contents of the bowel and the secondary fermentative or putrefactive changes taking place in them. As a result of the chemical or bacteriological irritation, inflammatory conditions and finally ulceration of the mucous membrane and submucosa occur. Formerly the direct mechanical irritation of retained contents, and especially of hardened masses, was considered of prime importance in the development of such ulcers, and the names, stercoral ulcer and decubital ulcer, were applied. According to Kocher, a better term would be distention ulcer, as he regards the ulceration as due to overdistention of the intestines, with consequent disturbance of circulation. When inflammatory changes are pronounced, the mucous membrane may be somewhat thickened, but nearly always it is thinner than normal, as the result of the distention.

The serous coat of the intestine is thinned by the distention, and more or less stretched in a longitudinal direction, as the result of a shortening of the mesentery by the dilated bowel.

Acute Obstruction.—When acute obstruction has followed a chronic but only partial occlusion, more marked distention of the bowel above the obstruction occurs as the result of suddenly increased pressure and interference with the movement of the intestinal contents, causing the accumulation of gases, in addition to the other intestinal contents.

In purely acute cases, there are quite marked differences from those met with in chronic obstruction. Below the obstructed area the bowel is empty and contracted, as in chronic cases.

Above the obstruction, the principal change is distention, with great thinning of the walls of the bowel. There is no evidence of hypertrophy, such as occurs in the chronic cases, owing, of course, to the acuteness of the condition. The contents of the bowel are, in the beginning, liquid or semiliquid, but after a relatively short time, gaseous accumulations occur, as a

result of interference with the circulation and consequent cessation of absorption from the mucous surface, and also as a result of the inability of the gases to pass the acutely obstructed portion. The distention is, in part, the result of the accumulating intestinal secretions and contents of the bowel, transported from above and stagnating above the point of obstruction. In part, no doubt, it is also a result of reflex nervous action, paralytic conditions of the bowel developing and favoring rapidly increasing dilatation. Such paresis of the bowel is greatly favored, also, by interference with the circulation and a hyperæmic condition of the walls of the gut. When complete stasis of the circulation has developed, absorption of accumulating gases ceases and increasing dilatation occurs.

The gases are partly derived from the air swallowed with food and in part from fermentative processes which become active on account of the obstruction. A considerable part of the distention may, however, be due to carbon dioxide derived from the venous blood of the stagnated intestinal circulation.

The condition of the bowel in strangulated coils is of particular importance. The obstruction of the circulation in such portions produces a rapid hyperæmia, and eventually a complete stasis of the circulation. The coil becomes of a deep red or purplish color, and is tensely distended, and hemorrhagic extravasation may occur within the lumen or on the outside. A gangrenous condition may develop, when the intestine becomes of a greenish or grayish color. Occasionally obstruction of the arteries, as well as of the veins, may cause a somewhat different appearance, the bowel in these cases remaining flaccid and becoming gangrenous in a very short time. The strangulated coil undergoes rapid distention from the accumulation of gases.

Affections of the peritoneum may occur in cases of strangulation, or in other forms of intestinal obstruction, due to the escape of microorganisms through the wall of the bowel in the area of obstruction, or immediately adjacent to it. The local conditions (compression of the tissues by bands, intussusception, etc.) and the complete stasis of the circulation cause degenerative changes in the wall of the bowel, and make them more readily permeable, and thus a rapid involvement of the peritoneum becomes possible. In cases of gangrene and perforation of the intestines, a violent local or perhaps general peritonitis quickly supervenes, and foul-smelling collections of inflammatory exudates and necrotic material, with escaped intestinal contents, are encountered.

The secondary changes immediately above the area of obstruction (venous stasis, paralysis of the bowel, and meteorism) increase the existing obstruction, and occasion the upward movement of the intestinal distention and accumulation at higher levels.

Symptoms.—The important clinical manifestations of obstruction of the intestines are pain, distention, nausea and vomiting, constipation, prostration, and collapse. A variety of secondary symptoms or variations in the principal manifestations occurs in the different types of the condition, and will be referred to in the discussion of the latter.

Pain.—This symptom differs greatly in manner of onset, severity, and character, according to the acuteness of the case.

In acute intestinal obstruction, sudden abdominal pain, usually of considerable violence, is the rule. This may be occasioned by the direct irrita-

tion of the nerves in the affected segment of the bowel, by the sudden compression or obstructive cause, and by the violent peristaltic efforts above the obstruction. As acute obstruction more commonly involves the small intestine than the large, the pain is more likely to be general than localized, as it is well known that the nervous mechanism prevents an accurate localization of pain involving the small bowel. In the majority of cases the pain of onset is referred to the middle portions of the abdomen. At first it is intense, and may continue so until, as a result of exhaustion, or possibly of toxæmia caused by the absorption of putrefactive products, a cessation is brought about. The pain may be so severe in the beginning that nausea, vomiting, or profound prostration may result. After an interval of cessation, secondary pains of cramp-like or colicky character due to violent peristalsis set in. These, however, are rarely prolonged, as are the pains met with in chronic obstructions. Very soon the bowel becomes fatigued, or prostration reaches a grade that puts an end to the patient's suffering.

Extension of infection to the surrounding peritoneum may add a new form of pain, sometimes distinguishable by its stabbing character and by its association with local tenderness and other symptoms. In other cases rapid abdominal distention may be the occasion of general abdominal pains described as of a rending character.

In chronic obstruction, pain may be wanting for a long time, and nearly always its onset is somewhat gradual. The patient first becomes aware of slight abdominal discomfort, due, apparently, to some interference with the movement of the contents of the bowel, and especially gases. Later, these sensations increase to a more definitely painful form; or, when there is a sudden increase in the degree of obstruction, intense, cramp-like paroxysms may set in. Not rarely the earlier symptoms have been so indefinite and uncertain that the occurrence of an entirely acute obstruction is suggested. The attacks of pain may become so severe that the patient screams or doubles up in agony. He is conscious of a more or less fixed location, and may feel the cramps begin at one portion and move more or less quickly to the place of obstruction, where the bowel becomes tensely distended, sometimes forming a visible hard mass. This indurated segment of the bowel may be so hard that on palpation it seems almost like a solid tumor. After a variable duration it relaxes, and relief may be obtained for a greater or less period of time.

Associated with the paroxysmal pains of chronic obstruction is a symptom of extreme importance in diagnosis, namely, visible peristaltic movements. Visible peristalsis may be seen through thin abdominal walls in persons not suffering with intestinal obstruction, but these movements are slow vermicular contractions, visible over a greater or less extent of the abdominal surface. They differ very decidedly from the intense and usually rapid waves met with in obstruction, and on palpation there is never the stiffening and evident tonic contraction found in cases of occlusion.

After a certain duration the colicky pains of chronic obstruction may subside, owing perhaps to fatigue or to relief of the local conditions. There may be an interval of hours, days, or even weeks before another attack sets in.

Metorism, or distention of the abdomen, is usually somewhat late in development, except in acute cases with strangulation and rapid interference with the circulation of more or less extensive segments of the bowel. In chronic cases there may be but little distention until near the termination of

the case, when complete obstruction has developed and paresis of the bowel above the obstructed area has caused interference with the circulation. Up to the very end the amount of distention may be relatively small.

In acute cases, with marked interference of the circulation, rapid and intense meteorism may cause early abdominal distention. In conditions of strangulation of coils of the intestine, paralytic distention of the strangulated area may occasion local meteorism of marked grade.

Constipation.—All forms of intestinal obstruction interfere with the onward movement of the intestinal contents. The resulting conditions, however, vary considerably in different cases.

In acute obstruction a sudden cessation of all intestinal movements may occasion an immediate absolute constipation, although frequently a movement or two may empty the lower bowel before the constipation becomes absolute. The higher in the intestinal tract the seat of obstruction is located, the more likely are some movements of the bowel to occur, after the development of the condition. The reasons for this are that the contents in the part of the bowel below the point of obstruction may be discharged after the obstruction has taken place, and also that the fluid contents of the upper intestine are more likely to pass a partial obstruction than are the semisolid or solid masses of the lower bowel to escape through an obstruction in that region.

In chronic obstruction, especially that involving the large intestine, constipation is sometimes absent or insignificant. In these cases the gradual development of the obstruction and the compensating hypertrophy of the musculature above the occlusion make it possible for the contents to escape in an approximately normal manner. Sometimes, however, considerable accumulations of fecal matter may be found above an area of obstruction in cases in which the movements of the bowels have seemed approximately or wholly normal.

Much importance has been thought to attach to the character of the stools in cases of partial obstruction of the lower bowel. Among other forms, small, separated, spherical masses and ribbon-shaped formations, or narrow, pencil-like stools, have been particularly noted. It is undoubtedly true that partial intestinal occlusions do occasion formations of this character; but exactly the same conditions may occur without any organic obstruction whatever, especially in cases of constipation due to local spasm of the bowel in cases of spastic contraction of the sphincter, etc.

The presence of blood, pus, and mucus with the bowel movements is not infrequent in obstructions due to organic conditions involving the lower bowel, especially in carcinoma and intussusception.

Diarrhœa.—Diarrhœa may sometimes occur in intestinal obstruction, instead of constipation, and may easily occasion a mistake in diagnosis. This is particularly liable to occur in chronic cases involving the large intestine, and is occasioned by the occurrence of inflammatory conditions of the mucous membrane above the obstruction. Some have believed that diarrhœa is always significant of ulceration, a view that is certainly not invariably correct. The diarrhœa may be continuous and chronic, or may be recurrent. The character of the movements varies, but there is usually a tendency to a considerable admixture of mucus, on account of the catarrhal conditions present. When ulceration exists, blood and pus may also occur in the movements.

Vomiting.—Soon after the development of intestinal obstruction, whether complete or only partial, nausea and disturbance of the stomach occur, and in many cases vomiting sets in quite promptly. In cases in which there has been marked pain, the nausea and vomiting are, in a measure, dependent upon the intensity of the suffering, and are clearly reflex. In such instances the sickness may be intense, and vomiting may be attended with violent straining.

The character of the vomiting varies at different stages. At first, the contents of the stomach are brought up, and sometimes, when the stomach has been overloaded at the moment of an acute obstruction, considerable relief follows the primary vomiting. Usually, however, vomiting continues without cessation, and after the food contained in the stomach has been discharged, watery fluid or bile is brought up. Eventually stercoraceous or faecal vomiting takes place, and is the most characteristic symptom. In the beginning the faecal odor is merely suggested. Soon, however, it becomes unmistakable. Finally, quantities of liquid vomit, of the foulest character, are discharged, sometimes being ejected with violence, while in other cases it wells up or gushes from the mouth.

A great deal of discussion has taken place regarding the causes of the faecal vomiting. Formerly various theories were entertained, such as those which attributed it to reversal of the peristalsis and to transportation of intestinal contents to the stomach from portions of the intestine just above the obstruction. There is no doubt that such antiperistalsis may occur, and that accumulated matter in the lower part of the small intestine may be brought to the stomach and vomited. Another theory was that of Brinton, who suggested that, while the ordinary peristalsis carries the contents adjacent to the walls of the bowel forward in the usual manner, a reversed axial current is taking place at the same time. Recent investigations regarding the whole subject, and a consideration of the functions of the different portions of the intestinal tract, would suggest that the symptom is due to the accumulation of secretions in the small intestine, and eventually an overflow. Physiologically, the small intestine is an actively secreting portion of the tract, while the large intestine is concerned with the absorption of fluid and the inspissation of the liquid contents received from above. Any obstruction in the small intestine, therefore, occasions an increasing accumulation of fluid contents, and eventually an overflow of stercoraceous material. Obstructions in the large intestine, especially when low down, may be unattended with this symptom from first to last, or accompanied by it only late in the course, because absorption still goes on in the portion of the large bowel above the obstruction, and prevents the overflow referred to.

Stercoraceous vomitus is usually a dark brownish or yellowish fluid, of an offensive odor, suggesting that of faeces. There are rarely solid particles, although such have occasionally been observed. Occasionally, stercoraceous vomiting may occur in gastro-intestinal diseases, unattended with intestinal obstruction, functional or organic. In these cases, bacterial decomposition of gastric contents, and especially of blood mixed with other gastric contents, may cause the suggestive odor. The organism most likely to produce this result is the *Bacillus coli*.

In cases of obstruction situated near the upper end of the small intestine, the vomiting may be characterized by the intense violence of the straining

efforts, by the comparatively small amount of matter brought up, and by the tendency to speedy collapse.

In cases of obstruction of the large intestine, and in chronic obstruction generally, vomiting is much less pronounced than in acute cases and those in which the small intestine especially is involved. Sometimes there may even be little nausea until a late stage has been reached, and the vomiting is usually rather closely dependent upon the taking of food, attempts at sitting up, or other movements or efforts. In the late stages, when violent attacks of colic have developed, vomiting of the same character as occurs in acute obstruction develops.

Prostration and Collapse.—In acute cases collapse may set in very promptly. In chronic forms of obstruction it may be absent until the final stages have been reached.

The most intense and sudden collapse occurs in cases in which the obstruction is situated high up in the small intestine. It is due to the sudden injury sustained by the nerves of the bowel, and is proportionate to the pain, retching, and other symptoms caused by the same nerve injury. It is, therefore, in the nature of a shock to the abdominal nervous system. There may be equally sudden prostration and collapse in obstructions situated lower in the small intestine; but more usually in these cases the patient becomes gradually weaker, as the result of continued nerve irritation, repeated vomiting, and loss of liquid from the system, until finally he passes into a state of collapse. In chronic obstruction, especially in cases of obstruction of the large intestine, collapse may be a very late development, and does not usually present itself until decomposition of retained products and toxæmia have occurred.

The appearance of the patient suffering with prostration and approaching collapse from intestinal obstruction is often characteristic. The skin is cold, pale, and generally bathed in sweat. The face wears an expression of anxiety and is pinched. The temples appear sunken. The eyes are depressed in their sockets and surrounded with dark rings. The respirations are shallow and often sighing. The temperature of the body falls. The pulse becomes rapid, feeble, and finally thready and irregular. The mucous membrane becomes dry, so that the mouth is literally parched. Urinary excretion diminishes until it may become suppressed. Finally, the patient sinks into a state of torpor, or complete stupor or coma. Before this stage is reached the increasing toxæmia and infection cause a cessation of all sensation, and the acute sufferings of the earlier stages of the disease subside. Sometimes the obtunding of sensory acuity develops without other manifest evidences of collapse, and may occasion a deceptive appearance of improvement.

The causes of collapse in intestinal obstruction are: (1) the primary shock to the nervous mechanism of the intestine; (2) repeated vomiting and loss of water from the system; (3) intoxication and infection. Sufficient reference has been made to the first of these causes. The loss of water from the system through vomiting and sweating may operate directly by reducing the strength of the patient, and indirectly by enormous inspissation of the blood. According to Nothnagel, the concentration may reach 24 per cent. of the total volume of blood.

The toxæmia of the late stages of obstruction is doubtless caused by a large variety of substances generated by the decomposition of retained

materials in the intestinal tract. As this form of decomposition takes place with greatest activity in the large bowel, toxæmic prostration and collapse are more marked in the cases in which the obstruction is situated in some part of the large bowel. Among the products of bacterial decomposition in the intestines that may be concerned in this toxæmia are indol, skatol, phenol, cresols, and sulphuretted hydrogen. All of these may play some part; but the number of possible decomposition products concerned in this condition may be very much greater than that named. Repeated vomiting and the consequent inanition may also lead to acid intoxication by the destructive decomposition of the fats of the body and the manufacture of acetone bodies or in other ways. Direct bacterial infection from the diseased portion of the bowel or from the adjacent peritoneum, when extension has taken place through the wall of the intestine, adds to the prostration of the patient and coöperates with the toxæmic causes of collapse.

Diagnosis.—General Diagnosis.—The existence of intestinal obstruction is usually rather easily determined, when the symptoms that have been mentioned present themselves in typical fashion. In acute cases the sudden occurrence of pain and the development of nausea and characteristic vomiting, the obstinate constipation, and the final prostration or collapse leave little doubt as to the nature of the condition. There are cases, however, in which, although the development of obstruction has been acute, some of these symptoms are distinctly less marked than has been described. On the other hand, there are conditions that must be discussed under the differential diagnosis, in which very similar symptoms are occasioned by diseases outside the intestine itself, although doubtless affecting the bowel in an indirect or reflex manner.

Differential Diagnosis between Intestinal Obstruction and Conditions Simulating It.—A variety of conditions may simulate intestinal obstruction more or less completely.

1. Various intra-abdominal conditions may occasion sudden intense pain, with nausea, vomiting, and collapse. Among such conditions are the passage of biliary or renal calculi, the rupture of gastric and duodenal ulcers, torsion of the pedicle of a movable spleen, crises in cases of movable kidney, pancreatic hemorrhage and acute pancreatitis, embolism or thrombosis of the mesenteric vessels, and nervous crises associated with locomotor ataxia or other nervous diseases. In all these cases the symptoms of onset may be similar to those met with in cases of intestinal obstruction. If, as a result of the violent irritation of the nervous system, a reflex paralysis of the intestine occurs, an actual intestinal obstruction may develop. This will be discussed under the head of Paralytic Obstruction of the Intestines. Usually, however, in the conditions named, although there is some weakening of motility of the bowels and consequently a certain degree of constipation, the obstruction is not absolute, and enemata rarely fail to bring away a certain amount of fecal matter. Distention of the abdomen, if it occurs, is only of moderate grade. The vomiting, although perhaps urgent in the beginning, does not persist as in the case of obstruction, and rarely becomes fecal in character.

2. In a second group of cases intestinal obstruction is suggested by the occurrence of absolute constipation. In a certain sense these cases are truly instances of actual obstruction, although the conditions occasioning the obstruction and the evolution of symptoms differ from those of in-

testinal obstruction in the ordinary sense. In this group may be included the paralytic condition of the bowel following pronounced forms of peritonitis. The onset of such cases, the early development of fever, the leukocytosis, and other evidences of infectious inflammation are important. The vomiting rarely becomes faecal in character, and constipation is not, as a rule, absolute. The marked local pain and tenderness distinguish such cases from intestinal obstruction, although secondary peritonitis may set in so early in certain forms of obstruction that the diagnosis becomes difficult.

In chronic peritonitis and tuberculous peritonitis, intestinal distention, constipation, and vomiting may suggest obstructed conditions of the bowel. The clinical course of these cases, however, is more prolonged; there is less vomiting than in cases of obstruction, rarely ever faecal vomiting; and the degree of constipation is rarely as great as in obstruction. Direct examination of the abdomen will usually detect local indurations, fluid exudate, or enlarged mesenteric glands.

3. In a third group of cases intestinal obstruction is suggested by the marked degree of abdominal distention, associated with some other, although less conspicuous, symptoms, suggesting obstruction. Thus in cases of infectious disease, such as typhoid fever, pneumonia, and violent acute intoxications, and infections of the bowel itself (enterocolitis), progressively increasing abdominal distention, together with nausea, vomiting, and constipation may cause a close simulation of certain forms of obstruction. In these cases, however, vomiting never becomes faecal in type, the degree of prostration and collapse is less pronounced, and the constipation is rarely absolute, so that the use of enemata usually suffices to obtain some faecal discharge. The clinical course of the whole case and usually the gradual development of the abdominal symptoms, the fever, and other indications of the primary disease serve to make the diagnosis easy in most cases.

In the case of certain nervous conditions, notably hysteria, marked distention of the abdomen (phantom tumor) may occasionally occur. In these cases there is often a striking lack of other symptoms, and the distention not rarely varies very quickly. Under psychic influences, and when the patient is asleep or is anaesthetized, such abdominal distentions may suddenly subside. Similar distention of the abdomen may occur in patients suffering with locomotor ataxia; but in these cases severe abdominal pain and sometimes associated gastric crises attended with vomiting are more apt to be present.

Diagnosis of the Situation of the Obstruction.—Sudden intense pain with early and marked nausea and vomiting is more characteristic of obstruction in the small intestine than of obstruction in the large. The pain is less definitely localized, the vomiting becomes stercoraceous earlier, and suppression of urine is a more marked and earlier manifestation than when the obstruction is situated in the large bowel, as the whole of the small intestine and part of the absorbing surface of the large intestine are in such cases left to exercise their normal functions. Obstructions in the large bowel usually occasion a more reliable localization of pain and tenderness, and the passage of mucous movements or blood and mucus are significant. When tonic peristaltic contractions of the bowel, with violent attacks of colic, set in, these may sometimes serve to locate the place of obstruction, as the wave of contraction may move indefinitely through the abdomen until it reaches the

point of obstruction, where it produces a stiffening from tonic spasm, and sometimes a hard tumor-like mass. This point may be recognized by the patient himself, and accurately located by him; or it may be seen through the abdominal walls or felt in palpating the abdomen. Obstructions in the lower part of the large intestine, especially in the rectum, may occasion ribbon-shaped or pencil-like stools for some time before the obstruction has become complete. Such movements, however, are not pathognomonic.

The Significance of the Urine in the Diagnosis of the Point of Obstruction.—The time of occurrence and the degree of indicanuria have been utilized as indications of the seat of obstruction. The early appearance of indican is undoubtedly of value in locating the obstructed point in the small intestines provided that other causes of indicanuria can be excluded.

The amount of urine has been referred to as sometimes significant. The higher the obstruction is situated in the small intestine the more intense will be the vomiting, and, consequently, the greater the loss of water from the system. At the same time, there is a failure of the absorption of liquid from the lower bowel, which occurs as in normal life when the obstruction is situated in the large intestine. Therefore, an early reduction in the quantity of urine is significant of the situation of the obstruction high up in the small intestine.

The differential diagnosis of the different forms of obstruction will be considered after these have been described.

Obstruction Due to Foreign Bodies or Fæcal Masses.—Among the causes of obstruction or obturation of the intestines included under this heading are gallstones, intestinal calculi and parasites, various foreign bodies, such as fruit stones, masses of vegetable or animal fibers or hairs, and masses of fæcal matter.

Gallstones.—Gallstones are a rare cause of intestinal obstruction, as their size is only infrequently sufficient to obstruct any portion of the intestinal tract. They may, however, aside from direct obstruction, be the occasion of volvulus or of obstruction resulting from adhesions between the gall-bladder and some portion of the intestinal tract.

This cause of obstruction was met with in 15 cases among 669 consecutive cases of intestinal obstruction at the London Hospital. The proportion of cases found by other authors, however, has varied from 1 in 15 (Fitz) to 1 in 28 (Leichtenstern). The condition occurs more frequently in women than in men, as the occurrence of gallstones is more common in the former than in the latter. The average age of the patients has been estimated at from fifty-seven years (Treves) to sixty-four years (Eve).

The gallstone usually finds its way into the intestine through a fistulous connection between the gall-bladder and the duodenum, ileum, or other portion of the intestinal tract. Less commonly, it slowly works its way along the common duct, ulceration opening the way before, and cicatrization contracting the channel behind the calculus. After reaching the intestine, the stone may cause obstruction of the duodenum, or it may move downward into the jejunum or ileum, and become impacted there. The most common situation of lodgement is in the lower part of the ileum, near the ileocaecal valve. Some cases have, however, been met with in which the stone caused obstruction of the large intestine.

After lodgement the stone may set up local irritation of the mucous membrane and ulceration of the bowel, with secondary peritonitis, and sometimes

rupture. Localized abscess formation and discharge of the stone through fistulous connections with the abdominal walls have been recorded. Occasionally, after primary obstruction, the occlusion may be relieved by the formation of a saccular diverticulum and the lodgement of the stone in this.

Symptoms.—The clinical manifestations may be divided into two stages: the first, that in which active symptoms result from the escape of the calculus from the gall-bladder or the biliary passages into the bowel; and the second, that in which intestinal obstruction occurs.

In the first stage, violent attacks of colicky pain attended with jaundice are characteristic. These symptoms may have been preceded by a clinical history suggestive of the presence of biliary calculi. After entering the bowel, evidences of partial and temporary, followed by complete, occlusion may present themselves. As the stone usually finds its way first into the upper part of the small intestine, the symptoms, such as vomiting and colicky pain, are comparatively severe. After a little while these may be relieved, but they may be renewed with each successive lodgement in positions lower down, until finally a complete impaction occurs in the ileum, and the evidences of total obstruction make their appearance. Even after complete impaction the calculus may become dislodged, and the symptoms, although intense, may be relieved and the stone discharged from the bowel. In cases in which relief does not occur, death usually takes place between the fifth and the tenth day, but sometimes later. The outlook is always grave, as approximately half of all the patients succumb.

Intestinal Concretions.—Intestinal concretions of sufficient size to produce obstruction are exceedingly infrequent. These concretions consist of organic substances, such as mucus, masses of bacteria, and fatty constituents, impregnated with calcium and magnesium salts. Such concretions are usually formed in cases of enteritis, and the symptoms are therefore preceded by those of this condition. Gradually, as the foreign body enlarges, the attacks of colic, diarrhoea, and other evidences of enteritis become altered and give place to the evidences of more or less complete occlusion. There may be marked tenderness, and even a tumefaction in the region of the obstruction. In case of the formation of concretions in pouches of the bowel, especially of the large intestine, evidences of partial obstruction are met with, and a continuous intestinal irritation or colitis is encountered.

Foreign Bodies.—Foreign bodies other than those described are comparatively rare causes of occlusion. Fruit stones, masses of intestinal parasites, hardened accumulations of mucus, balls of hair or vegetable fiber, and accumulations of medicinal substances, such as bismuth, salol, chalk, and other insoluble substances, taken by the mouth may occur. Obstruction due to inspissated faecal matter is referred to in the discussion of constipation. Such obstructions are at first partial, and may continue so for a long time. Hardened masses located in the lower part of the large intestine may set up catarrhal irritation and occasion a diarrhoea, which confuses the diagnosis. More commonly, faecal matter accumulates above and around the foreign body until more or less total obstruction has resulted. Simple faecal impaction and obstruction due to this are commonly the result of sluggish conditions of the bowel, such as occur in habitual constipation and in various diseases attended with general prostration. Not rarely the mass of hardened faecal matter can be felt through the abdominal wall, but the conditions of the

parietes, and especially the state of the muscles, may prevent accurate palpation.

Obstruction Due to Bands.—Intestinal obstruction may result from the constriction of a portion of the bowel by a band of adhesion formed by a previous inflammatory condition, or by various abnormal bands of fibrous tissue that result from developmental conditions. In a similar manner, a Meckel's diverticulum may be the occasion of this form of occlusion. Peritoneal bands, the result of previous attacks of inflammation, may stretch from some portion of the bowel to the abdominal wall or to various abdominal viscera. Subsequently, a coil of intestine may become constricted by passing beneath or over such a band, and the distention of the portion of the coil beyond the band may prevent its escape.

A Meckel's diverticulum is quite a frequent cause of obstruction, having been met with in 21 of the 669 cases at the London Hospital. If the diverticulum remains connected with the umbilicus, the conditions are such that obstruction of a coil of intestine may readily be produced; still more, when the free end of a diverticulum has formed a new attachment to the mesentery or some other part in the abdomen. A coil of bowel passing over or beneath the diverticulum may become twisted upon itself (volvulus), or may become variously knotted or contorted. Sometimes obstruction of the ileum occurs with a Meckel's diverticulum, as the result of stricture formation near the origin of the diverticulum, which is due to developmental conditions or traction.

Besides Meckel's diverticulum, bands may be formed by the attachment of the tip of the appendix to the appendices epiploicæ, the Fallopian tubes, or other structures within the abdomen, and strangulation of a loop of intestine may be the result. Occlusion by internal bands usually involves some part of the small intestine. The large bowel is relatively fixed in its position, and the lumen is such that strangulation beneath a band is infrequent.

A coil of bowel may be held beneath a band for some time without symptoms occurring. When, however, fecal accumulation or gaseous distention has increased its size, obstruction may occur, and may rapidly increase from pressure upon the veins and consequent stasis, which favors the development of still greater accumulation of gases within the occluded coil and prevents their absorption by the blood.

Symptoms.—The symptoms of intestinal occlusion by bands are usually sudden and acute, and frequently follow after some strain or effort, or after acute digestive disorders with distention of the intestinal tube. The symptoms are those of acute obstruction from any cause, beginning with sudden pain and marked nausea and disturbance of the stomach. The evidences of obstruction follow rapidly, and the patient suffers early prostration and collapse.

In many cases, however, the onset is less acute, and the symptoms are less violent. In these instances, pain and gastric disturbance may develop gradually, and distention of the abdomen increases progressively. Evidence of more or less obstruction may be found, but the nature of the obstruction may be quite uncertain. When a coil of intestine is strangulated by a band, the occluded portion tends to undergo pronounced distention from the accumulation of gases and the retention of its contents; and a painful swelling may be felt through the abdominal walls. The distention may be

so great that this is quite hard, and the percussion note over it may be relatively dull, from the tenseness of the walls.

Obstruction Due to Internal Hernia.—The term internal hernia is applied to hernial displacements of the intestines into the subperitoneal or retroperitoneal spaces without any protrusion externally. There are cases of external hernia in which no external tumor can be detected, although the hernia is strictly external, in the sense that the protrusion extends beyond the subperitoneal or retroperitoneal space through some abnormal opening toward the exterior.

Situations and Varieties.—Among the situations in which internal hernias may occur are: (a) The subperitoneal tissue in the region of the inguinal rings, when the hernia may be associated with external hernia or independent of the latter. Not rarely such hernias have resulted from the reduction en masse of an external hernia. (b) Duodenojejunal hernia, a protrusion of part of the jejunum into the retroperitoneal tissues on the left side of the abdomen, beneath the crescentic fold of peritoneum, the plica duodenojejunalis. A number of such instances have been reported, and occasionally strangulation has occurred. (c) Pericæcal herniæ, in which group are included hernias into the various pericæcal or retrocæcal pouches. (d) Intersigmoidal hernia, a form of considerable rarity, in which the hernial sac occupies the fossa occasioned by the sigmoid artery. (e) Herniæ of the foramen of Winslow, in which portions of the small intestines or, more rarely, of the large bowel extend through Winslow's foramen. (f) Herniæ into Douglas' pouch. (g) Herniæ projecting into the broad ligament of the uterus. (h) Diaphragmatic herniæ.

The last-named is the most frequent and most important form of internal hernia. Leichtenstern compiled 252 cases, and Grosser published anatomical descriptions based upon 433 cases reported in the literature up to 1899. Struppler found the number of cases reported up to 1901 to be 500.

A distinction is made between true and false diaphragmatic hernias, the former being cases in which the abdominal viscera extend through an opening in the diaphragm covered by a sac composed of peritoneum, pleura, or both. The false variety is that in which a free connection exists between the thoracic and the abdominal cavity. The latter variety is the more common of the two.

Diaphragmatic herniæ may be classified, also, as congenital or acquired, the latter being more frequent, and usually the result of traumatism causing rupture of the diaphragm. They are usually found on the left side, and among the positions most commonly involved are the opening for the œsophagus and an area immediately behind the stomach. The stomach is most frequently displaced through the abnormal opening, next to which in point of frequency, the colon, the omentum, or other parts of the intestinal tract may be involved. In exceptional cases, the spleen, the pancreas, or even the kidney is displaced into the thoracic cavity.

Symptoms.—Sometimes symptoms are wholly wanting. In these cases, a diagnosis may be made by the physical signs indicating the presence in the pleural cavity of air-containing viscera, and the restriction of respiratory movements on that side. Displacement of the heart may be more or less conspicuous.

The condition resembles most closely a pneumothorax, which, however, is usually distinguished by the fact that it commonly involves the whole

pleural cavity, and is frequently right-sided. The auscultatory phenomena, especially the occurrence of amphoric breathing when a fistulous communication with the pleural sac exists, are quite distinctive. Difficulty in swallowing, vomiting, and other gastric symptoms, and sometimes intestinal obstruction, may occur in diaphragmatic hernia, and are not met with in pneumothorax. Transillumination of the stomach has been practised for the diagnosis of diaphragmatic hernia, and skiagrams made after distending the stomach or colon with bismuth emulsions may be helpful.

Obstruction by Incarceration of the Bowel in Slits or Holes.—

An occasional cause of intestinal obstruction is the passage of a loop of the bowel through a hole or slit in the mesentery or omentum. The mesentery is the most frequent site of such imperfections; and Treves has described a spot between the ileocolic and terminal mesenteric artery, which is normally thin and readily becomes patulous. A gradual perforation of such a weak space may occur, but more commonly the perforation results from some violent injury that forces a portion of the bowel through the weakened area. Occasionally congenital holes are encountered. Sometimes, after inflammatory conditions, adhesions of the omentum and other structures may be so arranged that spaces are left between the adherent portions; and the bowel may be involved in these. The loop of intestine incarcerated by passage through an aperture may become more completely obstructed by twisting upon itself and thus becoming the seat of a volvulus. Obstruction may follow the incarceration of a portion of the bowel in a pro-peritoneal slit.

The symptoms of obstruction due to this cause are similar to those of incarceration by bands of adhesions. They develop with great acuteness, and usually are violent in their manifestations. No distinctive features are encountered.

Obstruction Due to Peritoneal Adhesions.—In this group are included cases in which the bowel is narrowed by peritoneal adhesions resulting from chronic peritonitis without strangulation of the circulation.

Etiology.—The adhesions that produce such constriction of the bowel may result from any form of peritonitis, such as that attending disease of the appendix, the pelvic organs, the mesenteric or other abdominal glands, or that associated with tuberculosis, cancer, etc. Similar adhesions may follow after operations, particularly when drainage has been required.

Pathological Anatomy.—The constriction may be annular, causing a limited contraction of the bowel similar to that due to stricture of the bowel, or a kinking or irregular contraction may be produced by looping over the bowel. In some cases the obstruction is due to a matting together of neighboring coils of intestine, or to close attachments to the parietal peritoneum by adhesions or by contraction of the mesentery. Annular adhesion or cicatricial formation in the peritoneum may surround the bowel completely like a ring, or may partially involve its circumference. Such constrictions may result from ulceration within the bowel, or may follow peritonitis, without involvement of the bowel wall itself. Annular constrictions of this type have been met with at the hepatic, splenic, or other flexures, causing close attachment of the bowel to the gall-bladder, liver, pylorus, spleen, diaphragm, etc.

Kinking of the bowel without any distinct annular stenosis may occur under a variety of conditions, in which, following a localized peritonitis, short and firm adhesions have formed between the bowel wall and the abdominal parietes or one of the abdominal viscera. This may occasion

an acute bend or kink, which, to all intents and purposes, produces the same result as that occasioned by a stricture of the bowel or an annular adhesion. In many instances such kinks are multiple.

In some cases, although adhesions between the bowel and neighboring structures occasion no sharp angulation, they may interfere sufficiently with the peristaltic action of the bowel, to result in partial obstruction.

A somewhat generalized peritonitis may be followed by a matting together of neighboring coils of intestine, with the result that considerable interference with the motor function may follow.

Symptoms.—There may be a noteworthy absence of all indications, and despite very extensive adhesions or deformations of the bowel, and sometimes, even in cases in which almost universal agglutination is met with, very little interference with peristalsis of the bowel is encountered. When obstruction occurs, the symptoms are those of gradually developing intestinal stenosis. As in other forms of organic obstruction, the symptoms may come on in an entirely gradual manner, or may develop rather suddenly, with attacks of pain, violent and visible peristalsis, and distention. Tumor-faction may be found at the seat of obstruction, owing to the meteoric distention of coils of intestine adjacent to or included within the zone of adhesion.

Obstruction Due to Stricture of the Intestine.—Definition.—By this term is meant the formation of a narrowing of the bowel by changes in its wall. This definition would include all forms of new-growths involving the bowel, as well as other disease processes affecting it. As here employed, it will be limited to cicatricial strictures, which in the great majority of cases follow ulceration. Tumors and some other conditions causing narrowing of the lumen of the bowel (diverticula) will be discussed elsewhere.

The various causes of ulceration of the bowel have been considered in another place. In this place it need be said only that, of these forms of ulceration, those due to tuberculosis, stercoral accumulations, ulcer of the duodenum, dysentery, and syphilis are the ones that are prone to be followed by cicatricial strictures. Considering the very large number of cases of typhoid fever that terminate in recovery, and the extent of ulceration in that disease, it is somewhat surprising how few occasion strictures. Nothnagel cites but two cases.

Stricture following tuberculous ulceration is much more common, the reason for this being that the process is a deeper one, runs transverse to the long axis of the bowel, and is frequently attended with hyperplastic processes that subsequently give place to fibrous tissue formation. Not rarely the strictures following tuberculosis are multiple, a number having sometimes been met with in one case. The hyperplastic form of tuberculosis has been considered in a separate place. In this form there may be marked encroachment upon the lumen of the bowel, just as in carcinoma or other tumors. In cicatricial ulceration of tuberculous nature, annular contractions occur without great thickening of the coats of the bowel.

Strictures following duodenal ulcer have the same general characters as the constrictions of the stomach or its pylorus resulting from gastric ulceration. Jejunal ulcers may have the same termination.

Strictures following stercoral ulceration are met with in various parts of the large bowel, especially at the flexures, in the cæcum, and in the rectum. An annular scar, with more or less thickening of the wall of the bowel, is usually

found in such cases. It is not improbable that secondary carcinomatous change occasionally originates in such lesions. Strictures following syphilitic ulceration are most common in the rectum. Marked thickening, with irregular scar formation and constriction, characterize these cases.

Strictures following dysentery were formerly regarded as very common. Woodward, in his collected statistics of the War of Secession, found not a single case of stricture in the 9431 fatal cases of dysentery occurring among the Union troops. A few instances have, however, been reported in literature, and Davidson states that he has seen 5 or 6 cases.

Strictures following various traumatic injuries of the bowel or after sloughing due to intussusception, hernia, etc., are occasionally met with.

Pathological Anatomy.—Cicatricial strictures of the bowel present the usual appearances of annular or irregular contractions of fibrous tissue, but are more or less modified according to the nature of the original ulceration. The majority of cases occupy some part of the large intestine, as it is in this part of the bowel that extensive and especially deep ulcerations most frequently occur. The greatest amount of thickening of the bowel wall is met with in syphilitic and tuberculous strictures. The condition of the bowel above the stenosis corresponds with that before described for all types of chronic intestinal obstruction.

Symptoms.—The symptoms of cicatricial stricture are those of stenosis. When a stricture occupies the small intestine, the symptoms may be relatively inconspicuous, owing to the fact that the intestinal contents in this portion of the bowel are normally fluid, and, therefore, unlikely to become stagnated. A stricture in the large bowel much more readily produces obstruction by the gradual accumulation of hardening fecal matter and the eventual development of complete obstruction.

Diagnosis.—The diagnosis offers greatest difficulty in cases of stricture of the large bowel, with hyperplasia of the intestinal walls. In such circumstances a differentiation from carcinoma may be very difficult.

Treatment.—The treatment in all cases is purely surgical, unless the stricture is of small caliber or is situated high up, when careful attention to the condition of the bowel may prevent difficulty.

Congenital Stenosis of the Intestine.—Congenital occlusion or stricture of the bowel is a not uncommon condition. The great majority of cases occur at the anorectal junction. Leichtenstern found the proportion of cases in the different situations as follows: 375 at the anorectal junction, 75 in the small intestine, and 10 in the colon.

Compression of the Bowel.—Intestinal obstruction is sometimes caused by the pressure on relatively fixed portions of the bowel by tumors, displaced organs, etc. Frequently localized peritonitis, with the formation of adhesions, contributes largely to the obstruction. The rectum is the portion of the intestine most frequently compressed, 60 per cent. of Leichtenstern's cases having this location. Among the causes of this condition, fibroid tumors, carcinoma, and misplacements of the uterus are the most frequent, but abscesses, extra-uterine pregnancy, and other causes are also met with. Compression may also occur in other parts of the large intestine, or even in the small bowel.

The *symptoms* are sometimes acute when the condition appears suddenly; but in the majority of cases some evidence of chronic obstruction precedes the acute symptoms.

Intussusception.—Intussusception, or invagination, is a condition in which one portion of the bowel extends into a neighboring portion in such a manner that the former is ensheathed by the latter. The part of the bowel within the ensheathing portion is known as the *intussusceptum*, the outer portion as the *intussusciens*. On transverse section through the bowel, including the ensheathing outer layer and the enclosed intussusceptum, there will be found three thicknesses of bowel: (1) the outer, or ensheathing; (2) the returning part of the intussusceptum; and (3) the entering part. The term *neck* is applied to that portion of the intussusception where the intussusceptum passes within the ensheathing bowel. The doubling of the bowel where the intussusciens joins the returning layer of the intussusceptum is known as the *collar*. Occasionally an intussusception is incomplete, lateral, or partial when only a part of the circumference of the bowel is drawn into another part.

An intussusception may be reducible or irreducible, incarcerated or strangulated. Finally, inflammatory changes, necrosis, or gangrene may take place. Intussusceptions may be either single or multiple, the latter being especially common in cases in which the condition attends the death agony.

Usually the intussusceptum extends downward into an intussusciens, but ascending or retrograde intussusceptions sometimes occur. The term compound intussusception has been applied to cases in which a second intussusception extends into the first, so that a cross-section would show five or more layers of bowel.

Etiology.—A great deal of discussion has arisen over the mechanism that occasions intussusception. As the result of this, it may be said that a distinction should be drawn between agonal intussusceptions and the ordinary pathological variety. In the former, which are multiple, violent peristaltic contractions doubtless play an important part. In Nothnagel's experiments, in which he tetanized portions of the intestines of rabbits, the tetanized segment became enclosed in the normal intestine beyond. The multiple intussusceptions of the agonal period probably result from similar violent peristalsis, often doubtless, irregular in character, and passing both upward and downward.

In the case of the pathological intussusception independent of the agonal period, the explanation is usually to be found in some organic defect, which occasions a prolapse of a segment of the bowel into a portion beyond; or in some pathological lesion of a part of the bowel which is carried downward into a portion beyond. Thus if a direct prolapsus of a portion of the bowel into a segment beyond occurs, the prolapsed portion of the bowel is seized by the peristaltic contractions of the part into which it extends, and is carried forward in the same manner as intestinal contents are usually transported. In the same way, a prolapsus or other tumor of the bowel, an inverted Meckel's diverticulum or appendix, or some other pathological condition may occasion the beginning of an intussusception. In the same manner, a prolapsus of the ileocecal valve is a common cause of the beginning of an intussusception.

Various investigations of the etiology of intussusception in cases in which no antecedent pathological lesion of the bowel has been found have indicated that the primary condition is a spasm of the circular muscle of the bowel in some portion. This constricted area becomes swallowed by the segment

of bowel immediately below, and thus starts the process that terminates in a complete invagination. A number of different developmental defects of the bowel may predispose strongly to this condition, and may be the initial cause, as when there is an undue size of the large intestine as compared with the small and a consequent tendency to prolapsus. Abnormalities of the mesentery similarly predispose, by permitting of the more ready invagination of one part of the bowel into another.

Immediate Causes.—The study of large series of cases of intussusception shows an absence of any definite immediate cause in half or even a larger proportion of the cases. In a relatively small number, some history of acute intestinal disturbance or of injuries, such as contractions, violent shaking, and the like, is met with.

Age.—Intussusception is the most common form of intestinal obstruction in childhood and infancy. Among Leichtenstern's 593 cases, 131 occurred during the first year of life. In Weiss' 321 cases, 177 occurred during the first year. In the latter series, 85 occurred in the second to the fourteenth year, and 59 after that time.

The Location of Intussusception.—The physiological and agonal invaginations, that is, those in which single or multiple invaginations have occurred as a result of irregular or violent peristalsis, are usually found in the small intestines. Pathological intussusception may occupy any part of the intestinal tract, but much more commonly originates at the ileocaecal valve than at any other point. In the majority of cases, especially in childhood, the ileocaecal valve prolapses into the caecum and thus becomes the apex of the intussusceptum dragging the ileum after it. The caecum and colon form the outer and middle layer, the ileum the inner. The term ileocaecal intussusception is applied to such cases. Less frequently the ileum prolapses through the valve into the colon and is carried farther on. The name ileocolic is given to this form, which is certainly rare. Leichtenstern found, in an analysis of cases at all ages, that invagination of the small into the large bowel occurred in 52 per cent., iliac intussusception in 30 per cent., and colic in 18 per cent. In adults the iliac and the ileocaecal varieties were of approximately equal frequency. Other large series have shown about similar proportions; but in small groups of cases great variations have naturally been encountered, which accounts for the varied opinions expressed by different writers.

Pathological Anatomy.—Secondary changes take place in the intussusception, according to the degree of constriction at the neck. There is practically always more or less interruption of the circulation in the mesentery entering into the invagination, and a consequent intense congestion of the intussusceptum. This becomes dark red, purplish, or blackish, and is swollen as the result of the hyperæmia and attendant oedema. Ecchymotic hemorrhages occur within the bowel wall, and exudation of hemorrhagic fluid into the lumen of the bowel takes place. The most marked change occurs in the middle layer of the invagination.

In the later stages, when the swelling of the invaginated portion has become extreme, complete obstruction of the lumen of the bowel occurs, and at the same time an increase of circulatory interference. As the result of this, gangrene ensues. In acute cases this may develop as early as the end of the first day; although it is usually not met with, even in the acute cases, before the expiration of three or four days. In the chronic cases gangrenous changes may be delayed for a long time, sometimes not appear-

ing until a number of months after the onset of the trouble. The gangrene usually first affects the apex of the intussusception, but in other instances it begins at the neck. As the result of the gangrenous necrosis, portions of sloughed-off bowel may be passed from the rectum. Sometimes these have been of very considerable length. In the celebrated case reported by Cruveilhier, it was three meters long. When the necrosis has affected the neck or some portion of the invagination near the neck, the discharged segment of bowel may be comparatively normal in appearance.

Peritonitis affecting the enfolded portion of the serous covering of the bowel (that is, the serous coat of the innermost and middle reflection of the bowel, where they lie in contact) sets in rather promptly; usually becoming marked by the end of the second or third day. This may be confined to the region of the neck of the invagination, or may be extensive, and involve the entire serous coating within the sac. As the result of peritonitis, the invaginated portions become agglutinated, and reduction of the intussusception more difficult. In the late stages, especially in the chronic cases, firm adhesions may form between the two layers of peritoneum adjacent to one another.

When such adhesions have formed at the neck of the sac, as the result of peritonitis, and the intussusceptum has been discharged after sloughing or gangrene, relief from the symptoms of obstruction may occur. More commonly a stenosis of the bowel results from cicatrization, and a partial intestinal obstruction, which later may become complete, is the terminal condition.

Symptoms.—The symptomatology varies widely, some cases being comparatively slow in development and chronic in course, and others highly acute in onset and terminating rapidly.

In the great majority of cases the onset is sudden, and occurs without any apparent predisposing condition. Sudden violent pain of a cramp-like character usually initiates the disease, and is speedily followed by nausea and vomiting. In many instances tenesmus and one or more movements of the bowels follow, and sometimes a diarrhoeal condition sets in. The movements may be composed of mucus alone, of mucus mixed with blood or of pure blood. The degree of tenesmus and of diarrhoea depends, to some extent, upon the situation of the intussusception, being more marked when this involves the lower portion of the bowel or the rectum.

Distention of the abdomen may appear, but is not, as a rule, a marked condition. Direct physical examination discloses a palpable tumor in many cases. This is usually felt in the left side of the abdomen, when the intussusception has become extensive and has reached that point. The tumor may, however, be felt at the ileocaecal region, where the intussusception, as a rule, begins. It is usually elongated, cylindrical, or sausage-shaped. Contractions of the bowel ensheathing the invaginated portion may cause a change in the consistency as well as in the size of the mass. Occasionally an intussusception extends so far as to protrude through the anus. In chronic or slowly developing cases the tumor may be found to change its position, moving in the direction of the intestinal tract.

In the acute cases, after an onset of this character, symptoms of depression and collapse occur, usually within from one to three days. At the same time the vomiting, as in other cases of intestinal obstruction, becomes faecal in

character. In chronic cases, if the onset has been acute, the symptoms gradually subside, and may become relatively inconspicuous. In other cases, the clinical course from the beginning is of a chronic character.

The most important symptoms are the pain, the tumor, and the peculiar behavior of the bowels. The pain in the early stages may be intense—so severe, indeed, that it occasions collapse. This is particularly marked in the acute forms of intussusception in early childhood. Later, paroxysms of colicky pain recur from time to time, with more or less frequency. Attending this, there may be a tonic contraction or stiffening of the segment of the intestine above the point of intussusception, as in other cases of intestinal obstruction.

Tenesmus is more frequently met with in this form of intestinal obstruction than in any other; it is earlier in its appearance, and more intense the nearer the invagination approaches the rectum. The character of the stools is a highly important diagnostic indication. There may at first be a discharge of faecal matter from the lower bowel, but soon the movements, if they continue, become mucous, bloody, or of mixed mucus and blood, while the discharge of faecal matter ceases. The evacuations may be attended with sharp recurrences of the characteristic tenesmus. Sometimes large hemorrhagic movements or actual hemorrhages from the bowel may be encountered.

Prognosis.—The termination of intussusception varies greatly. A rapid course with a fatal termination is more frequent in infants than in the intussusception of later life. Subacute and chronic cases, running a course of weeks or even months, occur, as a rule, in the older subjects. Even in these, however, the eventual termination is usually fatal. Reference has been made to occasional spontaneous cures; but, taking the number of these in large series, this favorable outcome is found to be exceedingly rare. Occasionally complete cure by spontaneous or induced disinvagination may take place in cases in which peritonitis and adhesions have not developed or have been so slight that the invaginated portion was able to escape. A few instances have been met with in which such relief from invagination occurred even after the condition had persisted for a considerable period of time.

Volvulus.—The term *volvulus* is applied to various kinds of twisting, knotting, or rotation of segments of bowel, resulting in partial or complete obstruction. A loop of intestine may become obstructed by rotating about its mesenteric attachment, which acts as a sort of pedicle; in other cases volvulus is due to a twisting or rotation of the bowel on its own longitudinal axis, while in still other cases one loop of intestine twines about the mesenteric attachment of another, or intertwining may take place between two or several coils.

Etiology.—Abnormal length of the mesenteric attachment of a loop of bowel, especially when combined with a lateral contraction of the mesentery, is the most important cause of volvulus. The mesentery may be elongated congenitally or it may have become stretched as a result of continued constipation and overloading of the affected portion of the bowel, and some stretching may result from adhesions of some part of the intestinal canal to fixed abdominal structures. A not uncommon cause of elongation is the inclusion of a coil of intestine in a hernial sac. This produces not only an elongation of the mesentery, but at the same time a lateral contraction, which

is not corrected when the coil is restored to the abdominal cavity. Narrowing or contraction of the mesentery may occur without special elongation as a result of inflammatory conditions, such as follow inflammation of the mesenteric lymphatic glands or other forms of inflammatory disease affecting the mesentery. A laxness and relative elongation of the mesentery may be caused by a loss of fat in the abdominal walls and in the mesentery itself. In women, after repeated pregnancy, these conditions frequently occur.

The conditions which predispose to volvulus are more frequently found in persons of advanced years than in the young, and volvulus is therefore most common in the aged or those past middle life.

The immediate cause may be a strain or some effort, as in lifting, or a traumatism, such as in compression of the body. Sometimes overloading of the intestinal tract combined with some effort or strain is of importance. Thus in old people who have suffered from habitual constipation the sigmoid flexure becomes elongated and its mesentery stretched. Finally an occasion arises when it is loaded with faecal matter and some strain occurs, as the result of which a volvulus develops.

Pathological Anatomy.—In simple volvulus, a loop of intestine twists about its mesenteric attachment. There may be a partial turn or several complete turns. In the former case the obstruction of the bowel is partial or incomplete; in the latter it is almost certainly complete, and in addition the bloodvessels become so compressed that intense venous engorgement results. Whether the obstruction is partial or complete, but especially in the latter case, distention of the obstructed portion of the bowel rapidly ensues. The bowel becomes dark red or purplish in color, and its walls are deeply engorged with blood and swollen by oedema. Then extravasations of blood are seen upon the surface and blood-stained liquid exudes into the lumen of the bowel as well as into the peritoneal cavity. Finally gangrene takes place. The distention of the obstructed coils, usually a striking feature, is due mainly to gaseous accumulation resulting in part from fermentative processes in the contents and interference with absorption of these gases owing to the stasis of the circulation and partly and perhaps largely to the accumulation of carbonic acid gas derived from the stagnant venous blood. The degree of distention sometimes becomes enormous, so that when the sigmoid flexure is involved the diameter of the affected portion may be several inches, and the distended sigmoid may fill the entire abdominal cavity and press the diaphragm far up into the chest. When a loop of the small intestine is involved the distention is so great that it appears at first sight to be a much enlarged colon. The striæ of the bowel are obliterated by the overextension, and rents of the serous coat may occur. Rupture of the bowel, however, rarely takes place. It is more likely to be found above the volvulus than in the affected portion.

Peritonitis sets in rather promptly as a result of the escape of bacteria through the injured, congested, and oedematous walls of the bowel. When bloody extravasation is present in the peritoneal cavity, the escaping bacteria may occasion putrefactive changes with the production of foul-smelling products.

Seats.—Volvulus most frequently involves the sigmoid flexure, but may occur at the junction of the small and large intestine or in some portion of the small intestine. In volvulus of the sigmoid there is usually a simple rotation

of the flexure about its mesenteric attachment. The rotation is usually in such a direction that the rectum is carried posteriorly to the descending colon, but sometimes the twist is in the opposite direction, so that the rectum is carried forward and lies in front. Occasionally the sigmoid may become coiled about a loop of the small intestine or about the pedicle of a pelvic or abdominal tumor.

Volvulus at the junction of the small and large intestine usually involves both the ileum and colon and is occasioned by an abnormal length of mesentery. Simple rotation of the colon on its own axis has been described, but is probably rare.

Volvulus of the small intestine resembles that of the sigmoid. Rarely two or more loops may be intertwined. Unusual length of the mesentery is the ordinary cause; but some other pathological condition, such as the passage of a loop of the bowel through an internal hernial orifice or under a band, may be the direct cause of the volvulus. In these conditions the loop of bowel after incarceration becomes twisted upon itself at the point where it is engaged in the hernial opening or under a band. A foreign body within the bowel may occasion the twist, just as overloading of the sigmoid flexure causes volvulus of this portion.

Symptoms.—The symptoms of volvulus are usually those of an acute intestinal obstruction. When the obstruction is only partial in the beginning, there may be a subacute onset which goes on to complete obstruction as the volvulus increases. The symptoms vary according to the location of the lesion. In volvulus of the sigmoid there is usually sudden severe pain followed by paroxysms of cramp or colic, and, as a rule, by absolute constipation. Reflex disturbances, such as vomiting, are often wanting, because of the low situation of the obstruction. Sometimes, instead of constipation, there is a tendency to mucous discharges with tenesmus, and occasionally ordinary diarrhoea may occur. The abdomen soon becomes intensely tender and enormously distended. Eventually this reaches a maximum grade and causes interference with breathing and circulation by upward displacement of the diaphragm. The superaddition of peritonitis, which usually occurs within twenty-four or forty-eight hours, causes increased tenderness and swelling and adds greatly to the prostration to which patients with this or other forms of acute obstruction are liable.

In volvulus affecting other portions of the intestine, the symptoms are similar; vomiting is more conspicuous, especially in cases in which the small intestine is involved in its upper portions.

In all cases the distention of the coil of the bowel included in the volvulus causes a localized tumefaction, which may be so hard that it simulates a solid mass. The situation of this may indicate the location of the volvulus, although it must be remembered that the length and mobility of a cæcum and even of the sigmoid may readily cause confusion.

Prognosis.—The prognosis of volvulus is usually very grave. In some instances the condition relieves itself spontaneously. This is probably more common in the case of the small intestine, but certainly occurs even when the sigmoid is involved. Usually, unless speedily relieved by surgical means, localized peritonitis and gangrene of the involved area soon take place; and death speedily follows.

Differential Diagnosis of the Conditions Causing Obstruction.—

Among the causes leading to acute intestinal obstruction have been mentioned: foreign bodies lodged in the bowel, compression by bands of adhesion, internal hernia, intussusception, and volvulus. Among the causes of chronic obstruction are various forms of stenosis or stricture of the bowel, cancer and other tumors, faecal accumulation, and compression of the bowel by misplaced organs, abdominal tumors, etc.

It is often impossible to distinguish between the different causes of acute or chronic obstruction, although sometimes antecedent conditions, the manner of development of the symptoms, and the clinical course of the case may furnish fairly reliable evidence of the nature of the condition.

Among foreign bodies, the most common cause of obstruction is gallstone. When a biliary calculus enters the duodenum and causes obstruction, the vomiting and prostration at once reach grades of the highest severity. Relief may follow the dislodgement of the stone and its transportation to lower levels; but if it remains high up, early faecal vomiting and collapse usually result. A preceding history of gallstone and jaundice or other significant symptoms may make the diagnosis fairly certain.

Obstruction by bands of adhesion usually involves some portion of the small intestine and causes symptoms of a decidedly acute character, with early development of faecal vomiting, prostration, etc.

Internal hernia also involves the small intestine, and cannot usually be distinguished with accuracy from the former.

Intussusception usually involves the ileum or the ileum and caecum, and is more frequently found in childhood or early life than at or after maturity. The symptoms of onset are decidedly less acute than in obstruction of the small bowel by bands. Localization of the place of obstruction is sometimes readily made by the situation of the pain, the development of a local tumefaction, and the situation of stiffening or tonic contraction of the bowel. The localization of the obstruction, the discovery of a sausage-shaped tumor, the characteristic stools, and especially the occurrence in childhood may establish the diagnosis. In acute cases, however, especially those occurring in young infants, the intense pain and sudden collapse may mask other indications, and the nature of the condition may be difficult to determine.

Volvulus usually involves the sigmoid flexure, and occurs in persons of somewhat advanced years, and especially in the aged. The symptoms are slow in development, and often inconspicuous until a late stage in the disease. The localization of the point of obstruction may often be readily established, although, sometimes a redundant sigmoid may occasion confusion.

Strictures and partial obstructions of the bowel may be suggested by the occasional occurrence of attacks of abdominal distention, with relief after active movements of flatus, but cannot generally be recognized with certainty.

Obstruction by carcinoma and other tumors usually occurs in the large bowel. Although tumors in their general relations are considered elsewhere, some of the features that indicate a neoplasm as the cause of intestinal obstruction may be appropriately referred to in this place.

Attacks of distention with active peristalsis and movements of flatus may occur from time to time, followed by more severe seizures, marked by violent abdominal colic. In these cases the patient himself may be able to recog-

nize the point of obstruction by feeling the place of spasmodic contraction above the lesions. On palpation, the physician may discover a hardening which appears at a certain place, and after a moment or two or a longer period of time relaxes. When the growth is situated favorably for palpation, or has reached considerable size, it may be discovered on abdominal palpation. When situated in the rectum, a digital rectal examination or the enteroscope may disclose its presence. As in other forms of obstruction involving the large bowel, the passage of stools of characteristic form some time before complete obstruction has developed and the occurrence of mucous, mucopurulent and blood-streaked or bloody passages may aid the diagnosis. Bloody and mucous movements are more frequent in carcinomatous obstructions than in any other form. The age of the patient, the gradual development of symptoms, and the occurrence of cachexia are important diagnostic features, but it must be recalled that some other conditions occasionally present a very puzzling resemblance. (See Diverticula.)

Obstructions due to faecal accumulation rarely occur independently of other causes of obstruction, except when the accumulation takes place in the rectum as the result of habitual constipation. In these cases the development of the condition is so gradual that the existence of obstruction may not manifest itself until, more or less suddenly, after the upper bowel has exhausted itself in efforts to maintain the onward movement of its contents, sudden paralysis takes place. In the earlier stages, the most characteristic indication is the increasing difficulty of securing evacuations and the insufficiency of these. With this, there may be a gradual increase in abdominal girth from general meteorism; and on careful palpation, the colon throughout its length may be found distended beyond normal dimensions. Hardened masses may also be discovered in the left iliac region, or, on digital examination, in the rectum. If the abdominal walls are thin and the peristalsis maintains itself actively, visible peristalsis may be seen for some time before the degree of contractions and the grade of obstruction cause sharp attacks of colic. It must not be forgotten that in many instances of gradually developing impaction, fairly regular movements, seemingly adequate in quantity, may occur for some time after the onset of the condition. Still more puzzling are the cases in which a continuous diarrhoea is maintained as the result of catarrhal inflammation of the bowel by retained faecal masses. A similar condition of things occurs in some instances of carcinoma of the lower bowel, with or without retention of faecal matter above the point of stricture.

Prognosis.—The termination of intestinal obstruction will, of course, depend upon the nature of the obstruction and its completeness. In acute occlusions that cannot be relieved death may occur within a few hours, or not until several or many days have elapsed. Leichtenstern estimates the average duration at six days. Partial or chronic obstructions may persist for very long periods of time; in some cases for years. Repeated vomiting, excessive shock, prostration, and collapse are the usual conditions that precede death in acute cases; the persistence of such conditions is, therefore, a prognostic indication of great gravity. Another cause of death in acute cases is peritonitis. In cases of greater duration, toxæmic manifestations usually set in before death ensues.

Recovery from intestinal obstruction may occur spontaneously in cases of faecal impaction or compression of the bowel; and sometimes in cases in

which the obstruction is due to adhesions, bands, internal hernia, or the presence of foreign bodies within the bowel. In intussusception, spontaneous disinvagination may occur, or the intussusceptum may slough off and recovery may take place, while in volvulus, the twisted loop of bowel may be spontaneously restored to its normal condition. A recurrence of obstruction after a primary relief sometimes takes place.

Treatment.—Practically all forms of intestinal obstruction demand surgical treatment. In cases of partial obstruction, however, and in some conditions like impaction of fæces or the presence of foreign bodies within the bowel, medical treatment deserves a careful trial. In the early stages, before an absolute diagnosis of obstruction has been made and before surgical intervention can properly be advised, certain indications must be met by medical means. In cases, however, in which it has been thought well to institute medical treatment, this form of management should not be persisted in longer than a day or two, if relief of the obstruction is not apparent. It is far better to err on the side of too early operation than the reverse; and in all cases of doubt, surgical rather than medical measures should be adopted.

In the beginning the patient should be placed at as complete rest as possible, and the diet should be restricted or completely withheld, even if marked gastric disturbances be not present. Small particles of ice may be allowed to quench thirst; but the unrestricted use of ice may occasion disturbance of the stomach, and is, therefore, objectionable. Enemata of water and hypodermic injections of saline solutions may sometimes be used to control the craving for fluid.

The use of laxatives, formerly employed in all varieties of obstruction of the bowel, must be condemned, except in certain special conditions. If they are effective at all in stimulating peristalsis, they will increase, instead of relieve, the abnormal conditions. Fortunately, when administered in error of diagnosis, vomiting usually causes their prompt expulsion.

In cases of fæcal impaction, purgatives are often desirable, although they must be employed with caution. Small doses of calomel or salines are preferable to more active remedies, because they produce serous outpourings without stimulating violent peristalsis. In cases of partial occlusion the accumulation of fæces may be prevented by the occasional use of the same or other laxative remedies.

Enemata and colonic flushings are especially advisable in cases of obstruction due to fæcal impaction. The repeated use of warm water, saline solutions, or oil may break up an impaction that threatens to cause complete obstruction. The injections must be given cautiously and without undue force, as the irritation caused by them may stimulate violent peristalsis above the point of obstruction. The same plan of treatment is sometimes effectual in dislodging a foreign body; but the use of forced injections of fluids in cases of intussusception, volvulus, etc., cannot be too strongly condemned. Formerly inflation of the bowel with atmospheric air or various gases was practised for the relief of intussusception and other kinds of organic obstruction. This method also merits condemnation.

Opium and atropine are administered with advantage in some cases of obstruction, for the purpose of quieting spasm or excessive peristaltic efforts. Thus in cases of internal hernia or occlusion by bands the obstruction may become more complete, as the result of the violent peristalsis stimulated at

the point of occlusion. Complete rest, the use of poultices or other warm applications, and small doses of opium or atropine, may relieve active peristalsis; and a release of the incarcerated bowel may take place, although this can rarely be hoped for. Similarly in cases of obstruction by foreign bodies the same remedies, especially atropine, may aid in relaxing spasm and thus facilitate the passage of the occluding body.

Besides these indications, opium or morphine is sometimes necessary, even though operation is decided on, to control the intolerable pain that attends acute obstruction. The doses should be no greater than are required to allay suffering; and, generally speaking, hypodermic injections are preferable to administration by the mouth or by the rectum.

External treatment applied to the abdomen may be useful in cases of obstruction which can be relieved, such as those due to foreign bodies or impaction, and some cases of incarceration by bands, through slits, etc. Large poultices or warm fomentations of various sorts may be used for this purpose. Even though ineffectual in accomplishing a relief of the obstruction, such applications frequently give comfort to the patient.

Massage of the abdomen is particularly helpful in cases of partial obstructions, such as are caused by adhesions after operations, or those due to the presence of foreign bodies. After relief from impaction in cases of narrowing of the lumen of the bowel, subsequent accumulations may be prevented by systematic manipulations combined with the administration of laxatives and the injection of warm saline solutions or oil into the bowel. In cases of acute obstruction, external manipulations must be used with the greatest caution, as any unnecessary stimulation of peristalsis cannot but be harmful.

Electrical stimulation of the bowel may sometimes be helpful in the same conditions in which massage is applicable, and it is to be condemned in the acute cases on similar grounds.

Certain symptoms may require treatment before or after operation has been decided on. Among these, gastric disturbances are most important. Nausea and vomiting may greatly complicate the difficulties under which the patient labors, and contribute largely to prostration. When laxatives are permissible, small doses of calomel placed upon the tongue may have a controlling effect. When the disturbance of the stomach is more intense, lavage of the stomach is advisable. If resorted to early, this method of treatment may reduce subsequent difficulties materially. Sometimes an actual cure of obstruction has been obtained from gastric lavage, as Kussmaul, who introduced this method of treatment, first pointed out. Surgeons quite uniformly recommended lavage as being useful in allaying the danger from subsequent anesthesia, when vomited material so readily gains access to the lungs. If used at all, lavage should be resorted to in the early stages.

Surgical Treatment.—In all cases of obstruction in which the symptoms are not relieved within forty-eight hours under general and medical management, surgical intervention must be advised, and in many cases immediate operation should be recommended. Attempts to relieve intussusception, volvulus, and complete obstructions by bands and the like by means other than surgical operation are inadvisable, although occasional cures have been obtained from such treatment. In the long run, the loss of time and strength thus occasioned makes the subsequent operative measures less likely to prove successful. It is outside the scope of the present work to

consider the details of surgical treatment, and a discussion of the forms of operation will, therefore, be omitted. With improved technique and the adoption of earlier operation, the results of surgical treatment have steadily improved. The mortality from obstruction, however, still remains a high one, despite all advances in knowledge and operative skill.

VOLVULUS OF THE OMENTUM.

Volvulus of the omentum may be considered briefly in this place on account of the resemblance of the symptoms to those of intestinal obstruction.

The great omentum may become twisted in the same manner as a coil of intestine. This usually takes place either in a part of the omentum included in a hernia or in a contracted portion that had been contained in a hernia and afterward restored to the abdominal cavity. Sometimes the twisting is the result of adhesions of the omentum to fixed structures. Occasionally several turns are observed, and some of these may be manifestly of considerable duration.

The omentum becomes swollen, œdematous, engorged with blood, and finally may undergo necrotic change from stasis. Hemorrhagic extravasations into its tissues and free exudation or extravasation into the abdominal cavity may take place.

Symptoms.—The symptoms are similar to those of intestinal obstruction. Sudden and severe abdominal pain with nausea and vomiting are the initial manifestations. The appearance of an abdominal mass, more or less tender on pressure, or the sudden distention of a hernial sac, if the twisted omentum is contained within the sac, are usually met with. Constipation is usually present but is less obstinate than in cases of volvulus of the bowel.

Treatment is purely surgical.

INTESTINAL OBSTRUCTION DUE TO MOTOR PARALYSIS OF THE BOWEL.

Partial or complete obstruction of the bowel may be due to motor insufficiency or complete paralysis of the motor power of the bowel. Sometimes this results from direct or reflex causes without organic lesions affecting the bowel itself. More frequently such obstruction from paralysis is a concomitant of organic diseases of the bowel.

Functional Paralysis.—A complete paralytic obstruction has occasionally been observed as the result of injuries or disease conditions, such as contusion, compression, or torsion of the ovary or testicle, various inflammatory conditions in the inguinal region or in hernial sacs, after operations for hemorrhoids, or in consequence of the passage of renal or biliary calculi. In such cases the paralysis of the bowel is a result of violent irritation of the sensory nervous mechanisms.

Cases have been observed in which the condition has followed paracentesis of large abdominal effusions or injuries to the abdominal cavity of various sorts, not involving the viscera. In a similar manner, after abdominal operations in which the intestines have not been involved, and in which

infection of the peritoneum has not been discoverable, a paralytic condition of the bowel has developed.

After the reduction of hernias, paralysis of the affected coils may occur, although the bowel presents no evidence of inflammatory or other organic change. In cases of intestinal obstruction involving the upper part of the small intestine, complete cessation of all movements indicates a secondary paralytic condition of the lower bowel.

In all these cases there is a functional paralysis without organic change in the wall of the intestine. This may be explained by a direct or reflex stimulation of the inhibitory nerve, the splanchnic, which occasions cessation of peristaltic movements. It has been held that in some cases, especially those following injury, operation or the reduction of hernia, an actual inflammation of the peritoneum is present, although it may be so slight as to escape detection. In some of the reported cases, however, careful investigation has excluded all possibility of even a microscopic inflammation of the peritoneum or of the walls of the bowel.

In another group of cases the paralytic condition of the bowel is probably due to toxic conditions affecting the intestinal walls or the nervous mechanism of peristalsis. In this group may be included the occasional cases of paralysis of the bowel occurring in the course of severe attacks of pleurisy or pneumonia. Mannaberg and the writer have observed instances of this sort. In some cases the obstruction was so severe as to call for operation. A similar condition occasionally attends cases of cholera, typhoid fever, and severe intestinal intoxications or enteritis. In all these instances the absorption of toxic matters acting upon the muscles or upon the nervous apparatus may explain the condition. It is possible that in cases of pneumonia and pleurisy a direct involvement of the splanchnic nerve may play some part. The absolute constipation of some cases of lead poisoning may be included in the group now under consideration.

Paralysis of the Bowel Attending Organic Disease.—Much more common than the cases of paralysis of functional character are the cases in which segments or a large part of the intestinal canal become obstructed by paralysis that is secondary to some form of intense organic disease. The most frequent and important cause of such paralysis is acute peritonitis. In cases of general peritonitis the bowel soon becomes quiescent; finally, all peristalsis ceases and a paralytic condition ensues. In cases of localized peritonitis, such as that attending appendicitis or, less commonly, other forms of inflammatory disease of the bowel, localized segments are involved. In chronic peritonitis and tuberculous peritonitis, obstruction of the bowel is more commonly due to adhesions or mechanical obstructions, although the inflammation of the bowel wall and the reflex nervous influences also play a part in the process. Embolism and thrombosis of the mesenteric vessels, by causing hemorrhagic infarction of the section of the bowel supplied by the occluded vessels, occasion a paralysis of this portion and consequent obstruction.

In all those cases in which paralysis has followed organic lesions the cause of the motor palsy is in part the direct involvement of the wall of the intestine in the disease process, and in part the effect of this condition upon the nervous apparatus.

Occasionally paralysis is due to excessive accumulations of intestinal gases or of fecal matter. In cases of chronic constipation or partial obstructions

of the bowel, or in various diseases in which a partial but not a complete paralysis has occurred, excessive meteorism may determine a complete paralysis; or the gradual accumulation of fecal matter may so distend the bowel as to cause the complete cessation of all peristaltic power. (See Idiopathic Dilatation of the Colon.)

Pathological Anatomy.—In cases of functional paralysis, the pathological anatomy offers little except the distention of the bowel. Sometimes, however, the paralyzed intestine is found contracted; and Leichtenstern, who has called attention to this condition, points out that in these instances there is simply an inhibition of muscular power, rather than an actual paralysis. When a limited section of the bowel is affected, as in cases of reduction of hernia, the bowel above the point of obstruction becomes more and more distended, while the affected segment may be flaccid and little distended, or, on the other hand, may be equally or excessively dilated.

In cases of paralysis following peritonitis, obstructions of circulation, or other inflammatory conditions, the anatomical features are mainly those of the condition that occasions the paralysis. The affected area of bowel tends to become distended with gas, and the bowel above enlarges progressively if the condition persists.

Symptoms.—The symptoms of paralysis of the bowel are practically those of obstruction by mechanical causes. When due to violent reflex nervous stimuli, as in cases of injury to the ovary or testicle, or to the passage of calculi, the onset may be attended with sudden intense pain, vomiting, and prostration, following which, obstinate constipation and distention of the abdomen make their appearance. Although vomiting is usually a marked symptom, it less commonly becomes fecal in character than in cases of mechanical obstruction. Other symptoms present in the cases may indicate the cause of the paralytic condition of the bowel, and may serve the purpose of establishing a correct diagnosis; although this is usually difficult.

The course of the condition depends upon the cause. In cases in which some injury has occasioned the paralysis, a return to normal conditions may occur after the original disease has subsided. Paresis of the bowel following operations, and that attending peritonitis, whether local or general, are exceedingly grave conditions.

Treatment.—Treatment must always be directed to the original cause of the intestinal condition. Sometimes this may be controlled by medical means, as in cases of toxic or inflammatory conditions of the bowels; in other cases, as when an injury has been sustained, local applications or cold or other local measures may be employed with advantage. Surgical intervention is frequently necessary.

Moderate paralytic conditions of the bowel may sometimes be corrected by hypodermic injections of sulphate of eserine (gr. $\frac{1}{100}$ to gr. $\frac{1}{30}$); by colonic douches of cool water or saline solution, or enemata containing stimulating remedies like asafoetida and turpentine. External applications, such as mustard plasters and turpentine stupes, may also be beneficial.

In cases of paralysis of the bowel following other abdominal diseases, surgical treatment of the original condition alone can be relied upon.

INTESTINAL DIVERTICULA.

Intestinal diverticula are classified as congenital and acquired. According to their anatomical peculiarities they can be divided into true and false diverticula. True diverticula are those composed of all the coats of the normal intestinal wall. False diverticula are those composed of only the mucosa and serosa. They are, in other words, hernial protrusions of the mucosa through defects in the muscularis. As most congenital diverticula are true and most acquired are false, the terms "congenital" and "true" and "acquired" and "false" have often been employed synonymously. Although in the majority of cases this holds good, some cases of the acquired form have apparently been true diverticula, so that the synonymous use of the terms "acquired" and "false" is not to be recommended.

Congenital Diverticula.—The so-called Meckel's diverticulum is the only form of congenital diverticulum that requires description. Other forms are of such extreme rarity as to be of no practical significance. Meckel's diverticulum results from the persistence of the omphalomesenteric duct. This should be entirely obliterated by the sixth or seventh week of foetal life, but some remains of it are found in from 2 to 3 per cent. of all cases. It is apparently more common in males than in females. Kelynack found 4 cases of Meckel's diverticulum in 298 autopsies, all 4 cases occurring in males, while in 337 cases Rolleston found 10 instances, 9 of which were in males. The diverticulum usually takes its origin from the convex side of the small intestine, about 1 meter above the ileocaecal valve. Exceptionally it arises from the upper portion of the small intestine. It varies from 3 to 10 cm. in length. Exceptionally it has been found as long as 25 cm. The wall of the diverticulum, commonly composed of the same structures as the intestinal wall, and its mucosa contain both Lieberkühn's glands and Peyer's patches. The lumen of the intestinal end of the diverticulum is large, frequently as large as that of the intestine itself. A valve-like fold of the mucous membrane at times occludes the opening of the diverticulum. Toward the free end of the diverticulum the lumen decreases, so that the process has something of a conical form. Occasionally the end is enlarged, at times assuming a hammer-like form. In unusual cases the same lumen is maintained throughout the entire extent of the diverticulum.

Usually the end lies free within the abdominal cavity. Occasionally, however, it remains attached to the umbilicus. Under these circumstances the lumen of the peripheral end alone becomes obliterated, forming a fibrous cord, but in other cases the entire duct may remain patulous, thus forming a free communication between the external surface and the ileum, permitting the discharge of intestinal contents at the umbilicus. This is a condition usually requiring early operative treatment. Hubbard has collected 9 such cases, in all of which operation resulted in a cure. The remains of the duct in the umbilicus may give rise to an adenomatous growth, as described by Kolaczek and Kustner. Greene has reported carcinoma of the same origin.

When, as is usually the case, the peripheral portion of the lumen becomes obliterated, the fibrous cord resulting may remain attached to the umbilicus and later become the possible cause of intestinal strangulation, or this cord may atrophy, and the end of the diverticulum then becomes free within the

abdominal cavity. Subsequently, however, inflammatory conditions may cause the free end to become adherent to any point within the abdominal cavity with which it may come in contact. This is most frequently the mesentery, but other points of attachment may be the parietal peritoneum, the omentum, the small intestine, the cæcum, or the colon. Cazin and Halstead¹ have made exhaustive analyses of the various places and structures to which the peripheral end of Meckel's diverticulum may attach itself, and Fitz² has thoroughly discussed the possibilities of strangulation of portions of the intestinal tract by such adherent diverticula. In exceptional cases, in which the lumen at the point of origin from the ileum has become obliterated or is closed by a valve-like fold of mucous membrane, a cyst may develop in the diverticulum and give rise to inflammatory phenomena or intestinal obstruction.

If, finally, the diverticulum assumes its most frequent form, a finger-like projection, the tip of which lies free within the abdominal cavity, it may remain an entirely harmless appendage throughout the patient's life. On the other hand, it may be the seat of various phenomena of extreme gravity. It may be the seat of a volvulus by rotating upon its own axis; it may be the starting point of an intussusception by becoming inverted into the ileum; or it may become knotted about a coil of intestines and produce stricture. If, as is sometimes the case, the diverticulum possesses a mesentery of its own, the latter may produce a stricture of the ileum. Mention has already been made of the possibility of the lumen becoming occluded at its intestinal end, inducing cyst formation which may suppurate or by pressure produce obstruction of the intestines. Ulcerations of the mucosa of the diverticulum may result from faecal accumulations or from typhoid or tuberculous infection. Occasionally such ulcers perforate. As all of the conditions of clinical importance to which a Meckel's diverticulum may give rise are treated of under their special headings, the reader is referred for their treatment to the appropriate chapters.

Acquired Diverticula.—Acquired diverticula may be true or false. True acquired diverticula are of extreme rarity. They are usually in the form of minute spherical or conical traction diverticula, occurring as a result of adhesions due to some extra-intestinal inflammatory condition. It appears possible, however, to have a true acquired diverticulum which is not the result of traction. Such a condition appears to have been present in the first case reported by Mayo, Wilson, and Giffin.³ False diverticula are hernial protrusions of the mucous coat through the muscular coat. As the protrusion of the mucous coat makes its way through the muscular coat it naturally carries the serosa with it, so that usually the wall of the diverticulum is composed of mucosa and serosa. At times a few muscular fibers are found between the two layers. These diverticula may be conical, cylindrical, hemispherical, or irregularly lobulated, and vary in size from that of a pea to that of an apple. They may occur throughout the entire intestinal tract, but are more frequently found in the large than in the small intestine. As a rule, they are multiple, and at times occur in extraordinary numbers. Hansemann has recorded as many as 400 in a case. They

¹ Halstead, *Annals of Surgery*, 1902, xxxv, p. 471.

² *American Journal of the Medical Sciences*, 1884, lxxxviii, p. 30.

³ *Transactions of the American Surgical Association*, 1907, p. 240.

differ from Meckel's diverticulum in that they usually arise from the concave side of the intestine close to the mesenteric attachment. At times they occur between the peritoneal coats of the mesentery. The factors concerned in the production of acquired false diverticula have been the subject of much discussion. It has been claimed that the simple intra-intestinal pressure resulting from long-continued constipation is the etiological factor. Another view is that they result from the atrophy of some of the fibrous elements of the intestinal wall. A view that at the present day has many adherents is that first advanced by Klebs, namely, that the point of entrance of the mesenteric vessels into the intestinal wall forms a focus of lessened resistance through which the mucosa forces itself, carrying with it the overlying serosa. On this basis it has been claimed that chronic passive congestion favors the formation of diverticula, but this claim does not seem to be substantiated by observation. From the evidence presented, the rational conclusion is that the primary factor in the production of diverticula is decreased resistance of the intestinal wall. This may be due to structural weakness at the points of entrance of the vessels or to areas of atrophy of the fibrous or muscular tissues of the bowel wall, probably of the nature of a process of senile involution, as the condition is met with most frequently in the aged. Constipation may be a factor in the production of the condition, by reason of the mechanical pressure, or possibly toxic action of accumulations of faecal matter between the folds of mucous membrane. The various theories of the production of intestinal diverticula are thoroughly discussed in an interesting article by Fischer.¹

According to Gordinier and Sampson, acquired diverticula were found 64 times in 8132 autopsies (Dresden Hospital); 19 times in 2600 autopsies (Johns Hopkins Hospital); twice in 953 autopsies (Bender Hygienic Laboratory); once in 2382 autopsies (Boston City Hospital). Careful investigation of left-sided tumors will doubtless reveal more.

According to Wilson,² infection in diverticula may sometimes affect the mucous lining, while in other cases penetration of the walls occurs, and a chronic inflammatory process surrounding the diverticulum results. He proposes the term diverticulitis for the former and peridiverticulitis for the latter. The former is primary in the mucosa, caused by bacteria to which it is not immune, causes no reduction of the lumen of the bowel, and tends to acute perforation into the peritoneal cavity. The latter is an inflammatory reaction of the peritoneum caused by a leakage through the walls of toxins or bacteria with which the mucosa is constantly familiar, causes a marked reduction in the lumen of the bowel, with chronic obstruction, and rarely causes perforation.

A variety of diverticula requiring special mention are those occurring in the duodenum at the site of the papilla of Vater. They are apparently due to weakening of the intestinal wall at the point of entrance of the bile duct. Keith claims that they occur in women from a forced visceroptosis, as the result of the pressure of corsets. The forced descent of the duodenum is not participated in by the bile or pancreatic ducts, on account of their more fibrous consistency, and a pouching of the intestinal wall in the neighborhood of the papilla thus results.

¹ *Journal of Experimental Medicine*, 1901, v, p. 333.

² See Mayo, Wilson, and Giffin, *loc. cit.*

Bland-Sutton¹ describes a form of abscess of the epiploic appendages caused by the penetration of foreign bodies through the intestinal wall and their lodgement in the epiploic appendage. Anatomically the appendix epiploica is a fold of peritoneum filled with fat, the latter being directly continuous with the subserous fat of the intestine. The base of the appendix in very fat individuals covers over a considerable arc of the intestine. Very often after middle life there appear in the wall of the gut, especially of the descending colon, small pouch-like diverticula, in which small foreign bodies may lodge and so penetrate the intestinal wall. In thin individuals the foreign body would probably enter the peritoneal cavity, while in the more obese it might enter the epiploic appendage and set up an inflammatory disturbance.

In a large majority of cases acquired diverticula give no clinical evidence of their existence. Lately, however, those situated in the descending colon, and especially in the sigmoid flexure, have been shown to be the cause of very profound diseased conditions, and their recognition explains some previously very obscure phenomena having their seat in the left iliac fossa. Among the contributions which have recently done much to shed light upon the condition are those of Mayo, Wilson, and Giffin, Brewer,² and Beer.³

The *clinical features* presented by these cases may conveniently be divided into the inflammatory and the obstructive.

The inflammatory phenomena may be the result of either a virulent infection of the diverticulum or perforation. The symptoms may briefly be described as those of appendicitis on the left side. The patients are usually males over forty-five years of age, otherwise in robust health, and generally inclining to obesity. The direct attack may be preceded by left-sided pain low in the abdomen, coming in spells and associated with constipation. Sudden, more acute pain, general at first, later localized to the left iliac fossa, and more or less paroxysmal in type, marks the onset of definite symptoms. Vomiting is usually not marked unless the pain is very severe. There is more or less rigidity of the left rectus muscle. The previous constipation may now change to diarrhoea. Finally a mass is discovered developing to the left of the median line in the middle or lower quadrant of the abdomen. In women this mass is at times pelvic rather than abdominal, due probably to the fact that the female pelvis is roomier and the sigmoid more movable. The general symptoms of infection depend upon the severity and extent of the inflammatory process.

In the obstructive cases the inflammatory symptoms are much less marked and the general symptoms of slowly progressive obstruction predominate. The condition may be readily confounded with carcinoma, especially as both conditions occur in advanced life; the formation of the tumor is, however, more rapid in the former. In diverticular obstruction there is usually a noticeable disharmony between the rather considerable size of the tumor and the lack of cachexia. Furthermore, the presence of blood in the stools would point strongly in favor of carcinoma.

The *treatment* of all forms of diverticula is purely surgical.

¹ *The Lancet*, 1903, vol. ii, p. 1148.

² *Transactions of the American Surgical Association*, 1907, p. 258.

³ *American Journal of the Medical Sciences*, 1904, cxxviii, p. 135.

CARCINOMA OF THE INTESTINES.

The malignant tumors of the intestines are carcinoma, sarcoma, and lymphosarcoma, of which carcinoma is by far the most frequent. Compiling the statistics of Maydl, Nothnagel, Heimann, Zemann, and Müller, we find that of 26,340 cases of carcinoma in general, 2255, or 8.56 per cent., were in some portion of the intestines inclusive of the rectum.

In determining the location of the tumor in the intestines there is a marked predominance of carcinoma of the colon and rectum. Combining the statistics of Maydl, Nothnagel, Zemann, Müller, and Bryant, it is seen that of the 659 instances of intestinal carcinoma that they have collected, 6.22 per cent. were in the small intestines, 6.82 per cent. in the cæcum and appendix, 22.76 per cent. in the various portions of the colon, and 64.18 per cent. in the sigmoid and rectum. The reason for this relative frequency of carcinoma in the colon, sigmoid, and rectum is rather obscure, unless it be that the mechanical irritation of the more or less formed faecal masses is the factor inducing the new growth. Another fact giving plausibility to the view that mechanical irritation is an important etiological factor, in the production of carcinoma of the intestine, is the relative frequency of carcinoma of the duodenum as compared with the ileum and jejunum, despite the great difference in the lengths. In the combined statistics of Maydl, Nothnagel, and Müller, of the 26 cases of carcinoma of the small intestine, 13 were in the duodenum and the remaining 13 in the ileum and jejunum. Rolleston states that he has collected 54 cases of primary cancer of the duodenum and but 19 of the jejunum and ileum.

Carcinoma of the intestine is in the vast majority of cases primary. Secondary involvement may occur either by metastasis or by direct extension. Metastatic involvement is seldom noted, and when it does occur it is not infrequently as a part of a general carcinomatosis. Metastatic carcinoma of the intestines is usually multiple. The nodules have a tendency to be located in the mesentery just at its attachment to the intestines, thus producing multiple stenoses without involvement of the mucosa. Direct extension of cancer of a neighboring organ to the intestines is somewhat more common and occurs especially as a result of carcinoma of the pancreas, gall ducts, and pelvic viscera. The question of the extension of carcinoma of the pylorus to the duodenum is an interesting one. Macroscopic extension to the duodenum is of rare occurrence. Cuneo and Lecène found that in only 1 out of 10 cases in which the pylorus was excised for carcinoma was there visible extension to the duodenum, but in 3 other cases microscopic examination revealed foci of carcinoma in the lymphatics, which, however, never extended more than 2 cm. from the pylorus.

Sex is of little etiological significance in cancer of the intestines. Males are apparently somewhat more frequently affected with carcinoma of the rectum and sigmoid, whereas this relative susceptibility is less evident in carcinoma of the upper portion of the tract. *Age* is probably a less important factor in carcinoma of the intestines than in carcinoma of almost any other portion of the body. The majority of cases occur in individuals over forty years of age, but not infrequently the condition is found in considerably younger individuals. According to Maydl, one-sixth of all cases of carcinoma of the intestines occur between thirty and forty years of age, and one-seventh

under thirty years of age. Bernoulli has collected 37 cases occurring in individuals under thirty years of age, and Nothnagel mentions 8 cases, 1 of which he himself saw, in individuals under twenty years of age. One of them was in a three-year-old boy, another in a boy aged three and a half years.

Pathology.—The majority of intestinal carcinomata are cylinder-celled adenocarcinomata taking their origin from the intestinal glands. Depending upon the relative proportions of the connective tissue to the epithelial elements, these tumors may be simple, medullary, or scirrhus. Of somewhat less frequent occurrence than cylinder-celled adenocarcinomata are cylinder-celled solid carcinomata and round-celled solid carcinomata, which may be either simple, scirrhus, or medullary; colloid cancer may occur in either the cylinder-celled or round-celled forms.

The macroscopic appearances that carcinomata of the intestines may assume are varied. As it is extremely seldom that carcinoma comes under observation during its earlier stages, only the more advanced forms will be described. It has already been mentioned that carcinoma of the intestines occurs much more frequently in the large than in the small intestine. The portions especially prone to be affected are the region of the cæcum, the hepatic and splenic flexures, the sigmoid, and the rectum. Frequently the tumor assumes a saucer-like form, having a necrotic ulcerated centre and elevated, indurated edges. Again, it is common to find the tumor extending about the entire lumen of the bowel in a girdle-like manner. Considerably rarer is the cauliflower-like mass of a papillary carcinoma. The tumors vary greatly in size. As the growth is dependent, to a great extent, upon the amount of epithelial tissue present, the larger forms are more commonly found among the medullary and simple carcinomata. However, on account of the larger size of these and their soft consistency, they are more prone to ulceration, and this process tends to diminish their size. The scirrhus forms, in which the connective tissue predominates, seldom assume very great dimensions, and on account of their density and small size they seldom undergo very extensive ulceration. They have a particular tendency to encircle the entire lumen of the bowel, and consequently stenosis is one of the most constant results. At times the fibrous elements so predominate in this type that the tumors have a cartilage-like consistency. Colloid cancers which are relatively most common in the rectum tend to infiltrate extensive areas of the intestinal wall. The surface usually shows considerable ulceration, the base of which is of a gelatinous consistency. On section they present this same appearance. The uninvolved portions of the intestines in the neighborhood of the carcinoma always show a marked grade of catarrhal inflammation.

Pathological Sequelæ of Carcinoma of the Intestines.—Obstruction.

—Both the degree and character of the obstruction differ materially in the various types of carcinoma and in individual cases. It may result in one of three ways. The carcinoma may produce an actual stricture of the bowel, as is most commonly seen in annular carcinoma, especially when of the scirrhus form. The same result may occur in the case of an annular carcinoma of a softer variety when in reaction to the carcinoma there is proliferation of the connective tissue of the bowel wall. In the second place, obstruction may result when the carcinoma so infiltrates the bowel wall as to materially increase the thickness of the intestinal wall and thus encroach upon the lumen. This is seen especially in colloid carcinoma, which, as has already

been mentioned, tends to infiltrate the entire circumference of the wall in the area of involvement. Lastly, obstruction may result when the carcinoma growing from the intestinal wall projects so far into the lumen as to occlude it. This form of obstruction is especially prone to occur in the case of simple and medullary carcinomata. In these types of tumor the condition results less commonly than would be supposed, for the reason that ulceration and disintegration of the tumor limit the proportion to which it would otherwise grow.

Stagnation of the Intestinal Contents and Dilatation of the Intestine.—These are conditions which are the direct results of the obstruction offered by the carcinoma. Not infrequently the stagnated contents just above the carcinoma become inspissated and hard, so that in palpating through the abdominal wall this mass may be mistaken for the tumor itself. This error probably accounts for those cases in which a large tumor is diagnosed, but in which one of small size is found on operation or postmortem examination.

Dilatation of the intestine consequent upon carcinomatous obstruction may be either acute or chronic. Chronic dilatation results in those cases of slowly progressive obstruction in which the bowel above the point of obstruction has time to accommodate itself in part to the obstruction. Consequently, in these cases more or less hypertrophy of the intestinal wall is always found in association with the dilatation. Acute dilatation may result in those rare cases in which the obstruction is of rapid formation, but is much more commonly found as a terminal phenomenon in chronic dilatation. Hypertrophy of the wall cannot go on indefinitely, and there naturally occurs a time when the pressure resulting from the force of the peristalsis above and the obstruction below overcomes the resistance of the bowel wall, and it balloons out. Occasionally in carcinoma of the colon, acute dilatation of the cæcum is found, although the portion of the colon above the obstruction is not dilated. The explanation of this peculiar phenomenon probably lies in the fact that the cæcum is subjected to the combined pressure of the peristalsis of the small intestines and the reverse peristalsis of the portion of the colon between the obstruction and the cæcum. Rupture of the intestines in acute dilatation may occur. For a more thorough consideration of the phenomena resulting from carcinomatous obstruction the reader is referred to the section on Intestinal Obstruction.

Adhesions.—In the majority of cases of carcinoma of the intestine of some duration, adhesions are found between the peritoneal covering of the area involved and some other portion of the peritoneum. They may bind the intestines to the abdominal wall, or bind coils of intestines to each other or to any of the abdominal or pelvic viscera with which they may come in contact. Frequently the adhesions are not limited to the region of the cancer, but involve areas for some distance surrounding it, and at times the adhesive process produces a fusing or matting together of the entire abdominal and pelvic contents.

Perforation.—Perforation into the general peritoneal cavity may occur when the destructive processes in the carcinoma are so rapid as to prevent the formation of adhesions. More common than this form of perforation is perforation into a walled-off sac of the peritoneum or into one of the hollow abdominal or pelvic viscera. In this case there is always first a binding down of the carcinomatous intestine to the portion concerned, and subsequently a perforation. The rupture and the resulting fistula may be between two

portions of the intestines, or between the intestine and the stomach, bladder, or uterus. Even perforation of the abdominal wall may occur so that a fistula is established between the intestine and the external surface of the body.

Peritonitis.—From what has been said above, it is seen that a generalized peritonitis may result when perforation occurs into the general peritoneal cavity or a localized peritoneal abscess form when perforation occurs into a walled-off portion of the peritoneum. Peritonitis, either general or localized, may, however, occur when there is no actual perforation. It results in all probability from increased permeability of the degenerated carcinomatous wall, permitting organisms to penetrate from the lumen of the bowel into the peritoneal cavity. Chronic adhesive peritonitis is included above under the discussion of adhesions.

Metastasis.—The commonest seats of the metastases from intestinal carcinoma are the lymph nodes, liver, and peritoneum. There is some difference of opinion as to the frequency of metastasis, especially from the large intestine. Müller, Hausmann, Maydl, and Rupp claim that metastasis to the lymph glands occurs late in carcinoma of the large intestines, and especially the rectum, while Hauser and others have found it early. The different varieties of carcinoma give rise to metastases more or less consistently to different regions. Medullary and simple carcinomata usually occasion metastasis to lymph nodes, especially to those in the region of the primary growth, while the scirrhus form is more prone to invade the liver, and colloid carcinoma shows a marked tendency to involve the peritoneum by direct extension as well as metastasis.

Symptoms.—As carcinoma in the different portions of the intestinal tract gives rise to essentially different groups of symptoms, the clinical features will be dealt with according to the location of the growth.

Carcinoma of the Duodenum.—The average age at which carcinoma of the duodenum occurs, according to the statistics of Rolleston based on 53 cases, is 51.6 years. It is rather more frequent in males than in females. Of 54 cases, 41 were males.

Carcinomata of the duodenum can be classified, according to their location, into those of the first or superior horizontal portion, those of the second or descending portion, and those of the third or inferior horizontal portion. Moreover, the symptoms usually differ materially according as the tumor is located in one or another of these portions. These differences are dependent primarily upon the seat of the tumor in its relation to the papilla of Vater, and as this is situated in the descending portion, the above classification holds good from the standpoint of the symptomatology as well as of the anatomical location. On account of this relationship between the seat of the tumor and the symptomatology, Boas has proposed the use of the terms suprapapillary, circumpapillary, and infrapapillary carcinoma.

Suprapapillary carcinoma presents in a general way the symptoms of carcinoma of the pylorus, especially those that are due to the purely mechanical effects of the obstruction in the latter. There is dilatation of the stomach with vomiting of the stagnated contents. At times the vomitus contains blood. Since the gastric mucous membrane is not involved in the carcinoma, free hydrochloric acid is usually present and consequently lactic acid and other products of fermentation are not present as they are in carcinoma of the pylorus. According to the Fenwicks, a tumor is palpable in 60 per

cent. of the cases. When palpable it is found to be less movable than carcinoma of the pylorus and is situated more to the right. Despite these points of differentiation from cancer of the pylorus, the diagnosis is always a difficult and frequently an impossible one.

Circumpapillary carcinoma or carcinoma of the second portion of the duodenum may present somewhat different symptoms depending upon whether the growth involves the papilla or not. If it does not, the symptoms are either those of carcinoma above or below the papilla. When, however, as is usually the case, the papilla is involved, more characteristic symptoms present themselves. The symptoms of pyloric obstruction may be present, but in addition there are the symptoms of obstructive jaundice. The stools are clay-colored, the tissues and urine deeply bile-stained, the liver enlarged, usually tender, and digestive and other disturbances dependent upon the obstruction to the flow of the bile and pancreatic juice present themselves. These symptoms, however, are usually not constant, for with the occurrence of ulceration the obstruction may be partially relieved and the escape of the bile made possible. Subsequently, however, the obstructive symptoms again present themselves, perhaps to be followed by another intermission. The ulceration gives access to an ascending infection of the biliary channels, so that a suppurative cholangitis may supervene, as indicated by an increase in the size and tenderness of the liver, chills, fever, and leukocytosis.

The severity of the gastric symptoms is dependent upon the degree of the stenosis caused by the cancer. If the growth should extend toward the pylorus and at the same time ulceration in the neighborhood of the papilla occur, the symptoms of suprapapillary carcinoma would supervene. Similarly, if the growth extended toward the third portion of the duodenum, and ulceration about the papilla occurred, the symptoms of infrapapillary carcinoma would present themselves. In many of the cancers in this location, and especially in those in which obstructive jaundice is the predominant feature, the differentiation from carcinoma of the ampulla of Vater or of the lower end of the common duct or of the head of the pancreas is extremely difficult. The occurrence of intermissions in the jaundice and the development of a suppurative cholangitis speak strongly for circumpapillary carcinoma. When a tumor is palpable it is felt as an immovable, hard mass deep in the right hypochondriac region near the midline. Either diarrhœa or constipation may be present, and naturally, in the course of time, anæmia and cachexia supervene.

Infrapapillary carcinoma characterizes itself by the symptoms of gastric obstruction associated with the vomiting of bile and pancreatic juice. The regurgitation of these fluids into the stomach neutralizes the acidity of its secretion, so that, although no free acid is found, the combined acidity may not be greatly decreased. The presence of the pancreatic secretion may be determined by its lyplotic action as well as its proteolytic action in alkaline media. The tumor is at times palpable as a firm immovable mass near the midline.

Carcinoma of the Ileum and Jejunum.—Carcinoma of the ileum and jejunum presents symptoms for the most part similar to those of the first portion of the colon. There is constipation or alternating constipation and diarrhœa. More or less definite symptoms of obstruction may present themselves, but these are not so frequent or marked as in cancer of the colon, on account of the more fluid contents of the bowel in the small intestine. Pain is one of

the most constant symptoms, and it is frequently of a colicky character. It is less definitely localized than is the pain of carcinoma of the large bowel. Occult blood is almost always present in the stools, and at times visible blood appears. Occasionally large hemorrhages occur. Although in some cases the tumor can be felt, this is by no means a constant phenomenon. When it can be felt it is characterized by its great movability unless it has been bound down by adhesions. On account of this great movability it is extremely prone to prolapse and is consequently not infrequently found in the lower abdomen or the pelvis. Cachexia and anæmia sooner or later make their appearance. As a rule, these symptoms are more marked than in carcinoma of the large intestine, for, other things being equal, the nearer a carcinoma of the intestine is to the stomach, the more profound are the cachexia and anæmia.

Carcinoma of the Colon.—Pain is a frequent but not invariable symptom of carcinoma of the large intestine. Some cases run almost their entire course, even to the development of a palpable tumor and marked cachexia without the occurrence of pain. The majority of cases, however, manifest some degree of pain during the course of the disease. It may vary from a vague sense of discomfort to a symptom of great severity, but seldom becomes extreme until obstruction becomes marked or absolute. Early in the condition the patient occasionally complains of diffuse abdominal pain without definite localization. Frequently accompanying this is a sense of pressure or dragging in the abdomen. Later the pain becomes more localized. Occasionally the pain is referred to distant parts, so that a patient with carcinoma of the cæcum may complain of pain in the left iliac fossa, or vice versa. This is a fact that should constantly be borne in mind in order to avoid errors in localization in case operation be undertaken. Another confusing phenomenon that is at times met with is referred pain extending along the course of the anterior crural or sciatic nerves and due to pressure on the intra-abdominal plexuses.

The variety of pain that is of greatest significance in the diagnosis of carcinoma of the intestine is colic. The attacks of colic are due to the progressive stenosis and the consequent distention and tonic peristaltic activity of the intestines above the stenosis. Not infrequently these paroxysms constitute the first symptom of which the patient complains. Generally the pain is diffuse throughout the abdomen, although occasionally the patient describes it as more or less localized. It may be that the patient refers the onset of these paroxysms to the eating of some indigestible article of food, to exposure or to some slight trauma, and in all probability these factors may play some part in the condition in inciting active peristalsis. At times a cathartic brings on the first paroxysm of colic. The pain may be felt by the patient to move from one position to another until it comes to an abrupt termination with a violent cramp, indicating the location of the obstructive growth.

A feature that practically always accompanies these attacks of colic is constipation. It is one of the most constant and frequently one of the earliest symptoms of intestinal carcinoma, and its occurrence in an individual past middle life should always excite one's suspicion. It is progressive in character, but it is occasionally interrupted by periods of normal activity of the bowels. These interruptions are sometimes due to ulceration of the tumor reëstablishing the lumen that had previously been stenosed, and con-

sequently such periods may be accompanied by the appearance of blood, mucus, and pus in the stools. Relief of constipation may, however, be due also to a more fluid condition of the intestinal contents at certain times. Again, periods of diarrhœa are by no means infrequent in the course of intestinal cancer. This diarrhœa is a natural result of the catarrhal inflammation that nearly always accompanies the carcinoma.

Tumor.—Of all the symptoms of carcinoma of the intestines, a palpable tumor is the most reliable and the most important. Its detection is not, however, essential to the diagnosis. Some cases run their entire course without the development of a palpable tumor at any time. In the majority of cases, however, careful and repeated examinations will reveal a mass in the course of the intestines. Naturally the case with which it can be determined will depend upon many factors, such as thickness of the abdominal walls and the presence of gaseous distention or ascites. The tumor may vary in size from that of a large nut to that of a child's head. In consistency it contrasts markedly with the surrounding abdominal contents, frequently feeling as firm as cartilage. It is round or irregular in form and its surface may be smooth or nodular. The infiltrating tumors, when they involve extensive areas of the bowel, give the impression to the palpating hand of a thick, solid cord. When a tumor is very small, percussion over it elicits a note not differing from that of the surrounding intestines. The larger tumors are usually dull to percussion. Pressure usually causes more or less tenderness, which is proportionate to the degree of spontaneous pain. The movability of carcinoma of the large intestine, unless it be bound down by adhesions, is one of its most characteristic features. This is especially true of carcinoma of the transverse colon and the sigmoid, but almost equally so of carcinoma of other portions of the large bowel.

A condition intimately associated with the tumor of intestinal carcinoma, and prone to cause confusion with it, is the tendency to fecal impaction above the point of obstruction. It is not unusual for this fecal mass to become so inspissated and compact as to cause the greatest difficulty in differentiating it from the carcinoma and lead to great misconceptions of the size of the growth. Usually, however, the consistency of this fecal mass is more doughy than that of the carcinoma, and unless the stenosis be complete, laxatives and enemata may speedily clear up the question. Other phenomena resulting from the stenosis that can at times be clearly observed are active peristalsis and distention of the portions of the bowel just above the carcinoma. They are, of course, both dependent upon the degree of the stenosis. In patients with thin abdominal walls, in whom hypertrophy of the intestines has occurred above the stenosed area, active peristaltic waves are frequently visible and at times serve as a means of localizing the growth. In carcinoma of the cæcum the peristalsis of the small intestine is seen in the central and lower portions of the abdomen, while in carcinoma of the lower portion of the colon these movements are seen in the course of the large bowel.

Gaseous distention, as a result of the stagnation of the intestinal contents, is at times so general that the portion involved cannot be determined by physical examination. In other cases, however, this distention is limited to the segment immediately above the carcinoma, and its observation is then of considerable value in the determination of the seat of the growth. If the distention is found to extend as far as the sigmoid, this would point

to the latter portion being the seat of the involvement, whereas if only the transverse and ascending portions of the colon are distended, the localization would in all probability be at the splenic flexure. In carcinoma of the hepatic flexure the ascending colon would be the natural seat of the distention.

The macroscopic and microscopic characteristics of the faeces occasionally furnish valuable information of the possible existence of a carcinoma of the large bowel. The form of the faeces is dependent upon the degree of the stenosis affected by the carcinoma. When the carcinoma is low down and the stenosis is of high degree, the faeces are generally of small diameter and either circular or ribbon shaped, or rudely rectangular in cross-section. Blood, pus, and mucus, either macroscopically or microscopically visible, are of themselves nowise diagnostic of carcinoma, but they are indicative of an ulcerative process in the intestines and consequently, in conjunction with other features suggestive of carcinoma, they present valuable contributory data. Blood, either visible or occult, and pus are derived from the ulcerated area itself, while the mucus is a product of the catarrhal inflammation of the neighboring mucous membrane. Blood may be present even when ulceration has not occurred. In such cases it proceeds from small erosions of the mucous membrane, sometimes the result of the mechanical irritation of the faecal masses. Pus is always an indication that ulceration has occurred. The association of pus and blood is of considerable diagnostic significance, for they occur together only in the most severe forms of intestinal ulceration. Shreds of tissue with the histological features of carcinoma may be present, and are, of course, of the greatest diagnostic significance. They are, of course, present only when ulceration has occurred.

Cachexia and anæmia are constant features of the later stages of carcinoma of the large bowel. They do not differ from the cachexia and anæmia of carcinoma in general, but are usually of later development in carcinoma of the large intestine than in carcinoma of the small intestine or stomach. The anæmia of intestinal cancer presents the type of an intense secondary anæmia.

Complications.—In addition to the above-mentioned symptoms, some or all of which are common to most carcinomata of the large intestine, various phenomena may occur which are the direct results of the carcinoma. Acute intestinal obstruction may occur at any time in the course of the disease. It may be the result of kinking by bands in the neighborhood of the carcinoma, or of volvulus or intussusception induced by the growth, or of the impaction of a foreign body in the partially stenosed lumen, or of the simple paralysis of the bowel above the stenosis. Peritonitis may result from rupture or from direct extension through the ulcerated intestinal wall. A localized peritoneal abscess may occur when adhesions have formed around the site of the peritonitis. Carcinomatous peritonitis resulting from the metastasis of an intestinal carcinoma to the peritoneum will be considered under diseases of the peritoneum. Perforation of a carcinoma of the large intestine into another hollow abdominal viscus occasionally occurs. Perforation of a carcinoma of the transverse colon into the stomach forming a gastrocolic fistula can be recognized by the presence of faecal vomiting and diarrhoea in which the stools contain undigested food particles. Perforation into another portion of the intestine may occur, and it is a peculiar fact that perforation of a carcinoma of the large intestine is more prone to occur

into another portion of the large intestine than into the small intestine. Perforation into the bladder can usually be recognized by the discharge of faecal elements with the urine.

Treatment.—When feasible, surgical treatment is employed, whether it be for removal of the growth or to overcome the results of stenosis. When surgical intervention cannot be resorted to, much can be done for the relief of the patient. The principal object to be attained is the prevention of faecal accumulation above the tumor. It is the mechanical and toxic effect of this accumulation that produces many of the symptoms from which the unfortunate patient suffers. Of the means that we have at our control to guard against it, the most important is the administration of such foods as contain a minimum of waste material, and in such form as to be most easily digested and absorbed. Small doses of laxatives may be frequently administered, and their action may be furthered by the occasional employment of oil enemata. Should there be much meteorism, intestinal antiseptics may be employed with advantage.

SARCOMA AND LYMPHOSARCOMA OF THE INTESTINES.

Although sarcoma and lymphosarcoma are histologically of entirely different structure, the nature of their involvement of the intestines and the resulting symptoms are so alike as to warrant their consideration together. Sarcomatous and lymphosarcomatous involvement of the intestines is much rarer than carcinomatous. Whereas, among the autopsies in the General Hospital of Vienna from the years 1882 to 1893, 243 carcinomata of the intestines were found, there were but 3 cases of sarcoma of the intestines and 9 cases of lymphosarcoma. In Bern 41 cases of carcinoma of the intestines were found to 1 of sarcoma. In contradistinction to the more frequent involvement of the large intestine as contrasted with the small intestine by carcinoma, sarcoma and lymphosarcoma involve the small intestine at least equally as frequently as they do the large intestine. Of 32 cases of intestinal sarcoma selected by Krueger, 16 were of the small intestine and 16 of the large intestine. In the 12 cases of sarcoma and lymphosarcoma mentioned above as occurring in Vienna, 8 involved the small intestine and 4 the large intestine.

The various portions of the small intestine are apparently involved with about equal frequency. In a series of cases collected by Libman, 15 were in the duodenum, 18 in the jejunum, and 14 in the ileum. In the large intestine the rectum is most frequently involved, and next in frequency the ileocaecal region. Jopson and White collected 22 cases of sarcoma and lymphosarcoma involving the large intestine.

Lymphosarcoma apparently takes its origin from the lymphatic tissue of the intestinal wall and has a great tendency to involve extensive areas of the wall by a process of infiltration. Sarcomata arise from the connective tissue of the submucous layer of the intestinal wall. Histologically, the most frequent form is the small round-celled sarcoma, but large round-celled, spindle-celled, and mixed forms may occur. These tumors also have a marked tendency to infiltrate extensive areas of the bowel wall, but at times they are more globular in form, when they may attain the size of a child's head. When they attain the latter proportions they frequently undergo

central softening, so as to give something of the impression of cyst formation. Sarcoma and lymphosarcoma may occur at any age from infancy on, but are most frequent in those under forty years of age. They are commoner in males than in females.

Symptoms.—The symptoms of sarcoma of the intestines differ materially from those of carcinoma. In carcinoma the local symptoms predominate, and they are frequently determined long before constitutional symptoms manifest themselves. In sarcoma, however, the local symptoms remain in the background, and usually attract attention long after the constitutional effect of the disease is noticed. This difference is due primarily to the fact that there is not the tendency to stenosis of the bowel in sarcoma and lymphosarcoma that is present in carcinoma. Sarcoma and lymphosarcoma invade the intestinal wall and early cause disintegration of its muscular elements, so that, instead of producing stenosis, they tend to cause dilatation of the involved area. Consequently, in the majority of cases there is marked wasting and anæmia before the symptoms point to any extent to the intestines. In a minority of cases the symptoms of obstruction may occur as a result of the growth of the tumor toward the intestinal lumen, or the destruction of the muscular elements of the wall may lead to paresis and consequent obstruction. Pain and diarrhoea are occasional symptoms. Blood, either occult or visible, may be found in the fæces, but aside from this the examination of the stools reveals no characteristic features. Occasionally fever as high as 101° and 102° is an accompaniment of sarcoma or lymphosarcoma of the intestines. Local examination will, in the majority of cases, reveal a tumor. Generally, this is in the nature of greatly thickened coils of intestines, but occasionally it is of a more circumscribed, globular nature, when it at times gives the impression of a thick-walled cyst. The duration of the disease contrasts with that of carcinoma of the intestines; in the majority of cases of sarcoma or lymphosarcoma of the intestines the patient dies in from four to eight months after the onset of symptoms.

Treatment.—Excision of the growth offers the only possibility of cure. Medical treatment can be directed only to the alleviation of symptoms.

BENIGN TUMORS OF THE INTESTINES.

Benign tumors of the intestines are of rare occurrence. In many cases they present no symptoms whatever, the first indication of their presence being their observation at autopsy. In other cases they give rise to various symptoms, but these are seldom characteristic enough to permit of a diagnosis during life.

The following varieties occur:

Adenoma.—Adenomas are the most frequent benign neoplasms of the intestines. The tumors take their origin from the glands of Brunner and Lieberkühn, and are usually either polypoid or papillomatous, more frequently the former. They vary in size from that of a pea to that of an orange. They are usually soft, but at times of firm consistency. On account of their rich vascularization, they are red and tend to bleed freely on manipulation. They are usually multiple. Luschka has reported a case in which he counted over a thousand tumors. This condition of multiple intestinal adenomas is termed by Hauser "polyposis intestinalis adenomatosa."

The tumors may be diffusely distributed throughout the entire intestinal tract from the pylorus to the anus, but are usually most numerous in the colon and rectum. In children, in whom they are relatively frequently found in the latter region, they tend to prolapse, and thus may become gangrenous or give rise to serious hemorrhages. Multiple polypoid adenomata of the large intestines have been especially studied by Quenu and Landal. Not infrequently these adenomas give rise to carcinomas; Quenu and Landal found carcinoma in 20 cases of 42 they studied. Weichselbaum observed a case in which a number of the polyps had undergone carcinomatous change. Childe reported carcinoma of the rectum in three sisters, in two of whom the carcinomas were secondary to multiple adenomas. According to Pellizari and Heurteux, in rare instances the pedicle of a polypoid adenoma has become atrophied and the tumor has been passed by the rectum. Multiple polypoid adenomas are most frequently met with in children from four to seven years of age. In 4 of the 13 cases reported by Post, the condition appeared to be hereditary, and Niewack has reported cases showing a family predisposition.

Lipoma.—Lipomas may be either broad-based or pedunculated, and either submucous or subserous. They may be small or as large as a child's head. When they assume the latter proportions they frequently produce obstruction; somewhat smaller growths may induce intussusception. They are found somewhat more frequently in the large than in the small intestine. Of 36 cases collected by Dewis, 19 were in the large intestine. The subserous lipoma frequently develops from the epiploic appendages. When pedunculated, the pedicle may atrophy and the growth may lie free in the peritoneal cavity. A submucous lipoma may become detached and may be expelled by the rectum. Instances of this occurrence have been reported by Castelain, Albrecht, Link, and Paci.

Myoma.—Myoma of the intestines is rare. It occurs oftener in the small than in the large intestine and may be internal or external. The former probably take their origin from the muscularis mucosa, the latter from the external muscular coat of the intestines. Steiner, in his exhaustive analysis of benign tumors of the intestines, reported 19 cases of internal and 15 of external myoma. They are more commonly found in the ileum and jejunum than in the duodenum and large intestine. These tumors may have a broad flat base and appear more as localized thickenings of the intestinal wall, or they may be pedunculated, thus taking the form of a polyp. Occasionally there is an extensive distribution of fibrous tissue with the muscular tissue, when the term fibromyoma is applied to them.

Hemangioma and Lymphangioma.—Hemangioma and lymphangioma are rare. McCallum collected 6 cases of hemangioma of the small intestine. They are usually cavernous as distinguished from telangiectatic hemangiomas; as a rule, they are small, flat, and multiple.

Symptoms.—Benign tumors of the intestines may exist throughout the patient's life without giving any indication of their presence. On the other hand, they may present symptoms of a more or less definite character, and at times give rise to conditions of great severity. In multiple intestinal polyposis, hemorrhage and diarrhœa are the most frequent symptoms. If the lesions are located in the colon and rectum, tenesmus may be associated. The stools frequently contain large quantities of mucus. As has been mentioned, the condition is most frequently found in children. A rectal

examination in a case presenting the above symptoms should never be neglected, as not infrequently in these cases the diagnosis will be made by finding numbers of the polyps springing from the wall of the rectum. In these cases the polyps are especially liable to constant irritation, and the presence of blood is a frequent symptom. If, as not infrequently occurs, these adenomatous polyps undergo carcinomatous degeneration, the symptoms of cachexia will later make their appearance.

In myoma, fibroma, and lipoma, hemorrhage and frequent stools, with mucus, may again be the predominant symptoms, but they occur much less frequently than in the multiple polypoid tumors above described. If the tumor is small and situated above the rectum or sigmoid, it usually remains undiscovered. If it is of larger size it may produce a slowly progressive obstruction, or, by inducing intussusception, cause acute obstruction. Steiner records 7 instances of intussusception in 18 cases of large intestinal myoma. When the tumor is of considerable size, it can frequently be palpated as a firm, nodular, rather freely movable mass. If it is in the lower part of the large intestine and extends downward it may be palpated or seen in the rectum. Angioma may give rise to extensive hemorrhages. It is impossible to make more than a presumptive diagnosis on the basis of intestinal hemorrhages in an individual previously healthy, and in whom no other cause for the condition can be determined.

Treatment.—The internal treatment of benign tumors of the intestines is limited to the treatment of the various symptoms. When, however, the symptoms are of sufficient severity or when the tumors are situated in the rectum and are consequently of easy access, surgical treatment is warranted. In case the tumor causes obstruction by occlusion of the lumen or by producing intussusception, operation must be undertaken. In an interesting case reported by v. Karajan, in which there were symptoms of obstruction and a number of palpable tumors, the intestines were opened in five places and eight polyps removed, the patient making a complete recovery. In case of hemorrhage of alarming severity from angioma or other benign tumor, operation may be employed. In those instances in which benign tumors are found in the rectum, even when they give rise to no symptoms, they should be removed on account of their tendency to undergo malignant change, as well as the possibility of their giving rise to dangerous hemorrhage.

EMBOLISM AND THROMBOSIS OF THE MESENTERIC ARTERIES.

Occlusion of the mesenteric arteries by embolism or thrombosis constitutes a condition of considerable clinical importance. In an analysis of 214 cases published by Jackson, Porter, and Quimby, in 1907, there were 197 instances in which an accurate study of the anatomical details was obtainable. Among these, 120, or 61 per cent., were cases of arterial obstruction, and 77, or 39 per cent., of venous obstruction. Embolism is considerably more frequent than thrombosis, the former condition having been present in 63 of 83 cases studied by Gallavardin.

Etiology.—Embolism of the mesenteric vessels is usually due to the lodgement of a portion of clot or fibrin dislodged from the endocardium, the valves of the heart, or from some portion of the arterial system above the mesenteric vessels. Endocarditis and atheromatous conditions of the

aorta or of the trunks of the mesenteric arteries are the important conditions antedating the embolism. The mesenteric vessels may be atheromatous without involvement of the aorta or of other vessels, and occlusion of smaller branches may be caused by emboli dislodged from the lining of the vessel near its origin. The embolus, finding its way into the smaller branches of the mesenteric artery, obstructs the lumen and produces various secondary results.

Thrombosis of the mesenteric arteries is liable to occur when there has been severe enteritis or infective conditions of the intestinal tract, as a result of which bacterial invasion into the bloodvessels takes place. Litten claimed that he had found thrombotic occlusion of the arteries as a consequence of diseased conditions of the arterial lining, but the correctness of his observation has been doubted. Syphilitic endarteritis has also been claimed as a cause of thrombosis, as has direct abdominal injury.

Venous thrombosis may occur as the result of cirrhosis of the liver, thrombosis of the portal vein, and other conditions causing stasis in the portal circulation.

Embolism and thrombosis of the mesenteric vessels occur much more frequently in men than in women. Jackson, Porter, and Quimby found 64 per cent. in men and 36 per cent. in women. Over half their cases were observed between the ages of thirty and sixty years.

Pathological Anatomy.—The superior mesenteric artery is affected much more frequently than is the inferior. The occlusion may involve the trunk of the vessel or one of the terminal branches. Sometimes it affects only the small terminals within the intestinal wall itself. In the last-named cases, small ulcerations of the mucosa are met with, as has been described in the section on Ulceration of the Bowel. The results of venous thrombosis are practically indistinguishable from those of arterial occlusion.

There may be a single occlusion of the trunk vessel, or multiple obstructions of small branches. In either case pathological changes occur in the mesentery and bowel embraced in the distribution of the obstructed vessel. The mesentery and the bowel become intensely engorged with blood, and assume a dark red and soon almost black color. Hemorrhagic extravasations take place into the tissues, and exudations of hemorrhagic fluid into the abdominal cavity and into the lumen of the bowel occur. The whole lesion is practically a hemorrhagic infarction of the affected area. The affected tissues may undergo rapid necrosis or gangrene, and perforative peritonitis may ensue. In the majority of cases a line of demarcation is observed. The affected bowel must be considerably distended with gas; and in some cases gaseous accumulation has been found in the peritoneal cavity, the result of the action of gas-forming bacteria.

The mucosa may present small ulcerations in cases of obstruction of the minute terminals within the wall of the intestine itself, while in cases of more extensive occlusion larger areas of ulceration of the mucous membrane may be found. Sometimes this takes a ring shape, surrounding the whole circumference of the bowel. The mesenteric glands may be greatly swollen, and the seat of hemorrhagic infiltration. The mesentery may be greatly thickened by extensive infiltration with blood, constituting a veritable hæmatoma.

The lesions are usually met with in the small intestine, which is supplied by the superior mesenteric artery. In the rare cases in which the inferior

mesenteric vessel is involved, the lesions are situated in the large bowel. Partial restoration, through the development of a collateral circulation, has been observed in a few instances.

Symptoms.—Two groups of cases are distinguishable, an acute and a chronic—the majority of the cases belonging to the former. In these there is sudden abdominal cramp or colicky pain, followed by nausea and vomiting. The vomitus at first consists of the contents of the stomach, but very speedily becomes hemorrhagic. Very soon diarrhœa sets in, and also becomes of hemorrhagic character. In other cases hemorrhage does not occur, and the symptoms are practically those of an acute intestinal obstruction caused by the paralytic condition of the affected portion of bowel.

The patient's temperature usually falls decidedly, he breaks into profuse perspiration, and frequently sinks into more or less profound collapse.

In some cases pain is wanting, and the earliest symptom may be a hemorrhagic diarrhœa. Pain, however, is usually a conspicuous symptom. In some instances the patient may suffer with repeated attacks of colicky pain, extending over a considerable period of time, without characteristic symptoms of vascular occlusion. In a case reported by Schnitzler, the patient suffered for six months with almost daily cramp-like pains, and with no other marked symptom except constipation. In the more acute cases, whether of the hemorrhagic type or of the form suggesting acute intestinal obstruction, intense abdominal pain of almost continuous character, or recurring in paroxysms, is met with. In over half the cases the pain is general. In the others it is limited to one or another region of the abdomen. Local tenderness on pressure may develop, and sometimes becomes extreme when peritonitis has set in.

Hemorrhagic diarrhœa is the most conspicuous and significant symptom, although it is not always present. The amount of blood sometimes becomes excessive; and usually there is rapid succession of movements each containing a considerable quantity of blood. When the hemorrhage is marked, the blood may be quickly passed and is only slightly altered. In other cases, black, tarry movements and a highly offensive odor indicate a longer retention within the bowel.

Distention of the affected area of the bowel sometimes develops rapidly, and general abdominal distention occurs in the cases in which intestinal obstruction is the important consequence of the vascular occlusion. In some instances a palpable tumor has been discovered in the abdomen, the mass being caused by the infiltrated mesentery and bowel.

In the more chronic cases, which include most of the instances of venous thrombosis, as well as some cases of arterial occlusion, the onset may be insidious and the symptoms of a remittent type. At times there are no abdominal symptoms of any sort; or, at most, vague and indefinite manifestations.

The clinical course of the condition may be exceedingly rapid. Jackson, Porter, and Quimby found, in their analysis, that 20 per cent. of the cases of either arterial or venous occlusion had a duration of but twenty-four hours; and 18 per cent. and 22 per cent. of the venous and arterial cases, respectively, a duration of but two days. In 8 and 10 per cent., respectively, the duration was three days. Of the prolonged cases, those due to venous closure showed gradual and continuous progression, while the arterial cases were more apt to present interrupted attacks.

In the very acute cases, after the initial pain and vomiting, rapid distention of the abdomen, cessation of peristalsis, and speedy death, following symptoms significant of absolute intestinal obstruction, mark the course of the disease.

Diagnosis.—The rules laid down by Gerhardts are still followed with advantage: (1) There must be a source of embolism. (2) Copious intestinal hemorrhages unexplained by organic disease of the bowel or by portal obstruction. (3) A rapid and marked fall of the temperature. (4) More or less severe colicky abdominal pains. (5) Distention of the abdomen and the accumulation of free abdominal fluid. (6) The occurrence of embolism elsewhere, before or simultaneous with obstruction of the mesenteric vessels. (7) The discovery of a palpable mass (mesenteric hæmatoma).

Portal obstructions without thrombosis may be indistinguishable from those in which thrombosis has supervened. The difference is merely one of degree.

Prognosis.—With few exceptions, the disease terminates fatally. In the statistics before quoted there are recorded 14 cases in which the diagnosis seemed reasonably accurate and in which recovery ensued. This favorable termination can occur only when a collateral circulation is speedily established.

Treatment.—The treatment of this condition is purely surgical. Jackson, Porter, and Quinby refer to 47 cases in which an exploratory laparotomy was undertaken. The mortality in these cases was 92 per cent. Four patients recovered.

ARTERIOSCLEROTIC INTESTINAL DISTURBANCES.

Certain painful conditions of the intestine are occasionally met with in the aged and arteriosclerotic, and are of some importance on account of their simulation of organic or inflammatory disease of the bowel. The onset may be abrupt, like that of acute intestinal disturbance, and pain may be severe. Usually there is constipation; and sometimes this is obstinate. Later, and especially after the use of laxatives, diarrhœa may set in. Localized soreness or tenderness may be discovered, but there is rarely marked rigidity of the muscles. The nature of these conditions is uncertain. In some cases, doubtless, obstructive conditions of the circulation play an important part. In a case under the observation of the writer, after a number of attacks, death occurred from a severe paroxysm with obstinate constipation, which was followed by the development of uræmia. At autopsy a partial obstruction of a branch of the superior mesenteric artery was found; and the section of bowel supplied by this was intensely engorged with blood. There was not complete obstruction, and no actual hemorrhagic infarction. Ortner has referred to cases somewhat resembling this, under the title of Intermittent Angiosclerotic Dysphagia of the Intestines. He suggests the resemblance to intermittent claudication, and refers particularly to the intermittent character of the symptoms.

MUCOUS COLITIS.

Definition.—The term mucous colitis is applied to a condition in which the patient suffers with various symptoms of disturbance of the stomach and bowels and with the regular or periodic discharge of masses of mucus or

mucomembranous shreds or casts of the bowel. The terms *membranous colitis*, *tubular diarrhœa*, *mucous colic*, and *mucous diarrhœa* have been applied, and sometimes, although with insufficient justification, a distinction has been drawn between these as being different forms of disease. It is possible to distinguish cases in which mucous or membranous diarrhœa occurs as an independent affection and those in which it is secondary to some other disease of the bowel, but there is no fundamental difference between the cases in which a discharge of masses of gelatinous mucus occurs and those in which membranes or casts of the bowel are met with.

Etiology.—The disease is most common in the middle period of life—that is, between thirty and fifty years; but it sometimes occurs in children or those below thirty years, and occasionally persists after fifty years. It is more common in women than in men, the proportion probably being 5 or 10 to 1. The disease affects a person of a better station of life more frequently than the poor, and is commonly associated with a nervous temperament or with distinct nervous disease. Not rarely it develops in hysterical or neurasthenic women, particularly if they have suffered with dyspeptic symptoms and constipation. It may follow after other forms of intestinal disease, the secondary variety being a frequent accompaniment of such diseases as chronic appendicitis, partial intestinal obstruction, dysentery, tumors, and long-standing constipation.

The direct cause of the disease probably varies greatly. Nutritional conditions such as are met with in those of reduced strength and those having nervous diseases, seem to play a predisposing role. Some authors regard the condition simply as an intestinal neurosis with excessive mucus formation (*myxoneurosis*). Recently it has been claimed that certain bacteria play a part in the causation, among other organisms being the *Bacillus coli communis*. A variety of membranous colitis, probably properly distinguishable from that now under consideration, occurs in association with conditions of general infection or septicopyæmia. In these circumstances a more or less widespread membranous colitis of diphtheritic type may occur.

Pathological Anatomy.—The characteristic feature of this condition is the formation of excessive quantities of mucus. These may occur as masses or balls of gelatinous character, which may be discharged as such or mixed with brownish, faecal matter of liquid, semiliquid, or even solid consistency; or, more characteristically, the mucus may attach itself to the wall of the bowel in the form of more or less tenacious and laminated membranes, which may subsequently be discharged as skins, shreds, long tape-like bands, or even casts representing quite accurately a mould of the bowel. Probably the commonest form is that in which the skins or shreds of membrane are discharged in masses or mixed with soft or liquid faecal matter. Not rarely these present the appearance of segments or longer portions of tape-worms, and are quite commonly mistaken for these by the patients. On careful examination, the shreds or casts are found to be somewhat laminated, and may show depressions corresponding with the openings of the glands of the mucous membrane. Epithelial cells may be found clinging to the outer surface. The mucous membrane itself may present an inflamed appearance, but usually there is surprisingly little evidence of any structural disease of the bowel. Often an atonic dilatation of the colon is observed, but in other cases a firm toxic contraction of the bowel causes a narrowing of the lumen and an apparent thickening of the wall. In many cases there is an association of hemorrhoids.

Symptoms.—The symptoms met with in this condition are gastric, intestinal, nervous, and nutritional. Usually the patient complains of some form of indigestion or dyspepsia. The appetite is poor and capricious; and the patient accustoms herself to greater and greater restriction of diet, as the result of attempting to find a dietary that will not cause discomfort. Distention of the stomach, flatulence, heaviness, or pain after eating, nausea, and other gastric symptoms may occur. Sometimes pronounced manifestations of various forms of nervous dyspepsia are present.

Among the intestinal symptoms, constipation and the discharge of characteristic mucus- or membrane-containing stools, are most important. The majority of patients are constipated, and the discharge of mucus or membrane occurs from time to time, attended with painful paroxysms of varying grades of severity. In cases in which large casts or abundant membranous shreds are passed, an attack of abdominal pain of great violence may precede this, suggesting some form of intensely acute inflammatory trouble. After the passage of the mucus or membrane the painful attack ceases, but a rawness or soreness of the bowel and abdomen is left which may persist for some time. In the majority of cases, however, such painful paroxysms are exceptional, and not rarely the passage of the mucus or membrane is accompanied by a relief of various symptoms that have previously been in evidence, such as intestinal distention, general abdominal discomfort, depression, low spirits, etc. Occasionally instead of constipation, the patient suffers with continued looseness of the bowels and the passage of large quantities of mucus mixed with fecal matter.

On examination, the abdomen is found distended or scaphoid. Preceding attacks, there may be increasing distention of the colon, which later gives place to a contracted or scaphoid condition; but in many instances distention is wanting at all times, and on direct examination the colon is found contracted as a hard tube. It may be possible to palpate the ascending and descending colon and the sigmoid flexure throughout the greater part of their course. The transverse colon is less commonly palpable.

The patient may for a long time suffer with gastric and vague intestinal symptoms and constipation, and may become increasingly nervous, before the characteristic symptoms of mucus- or membrane-formation develop. In such cases the mucous colitis is probably a secondary development.

Sometimes when shreds of membrane or casts are passed there may be a certain amount of blood streaking the membranes; and in exceptional instances a considerable quantity of blood may be discharged.

The nervous manifestations of the disease are usually striking. It is difficult to determine to what degree these are the result of the condition, rather than the predisposing cause of it. Nearly all patients that suffer with this condition are depressed, gloomy, irritable, and of low vitality. Distinct hypochondriacal tendencies may be marked, and sometimes the patient becomes actually melancholy. Reflex nervous disturbances of various sorts may occur; such as weakness and irregularity of heart action, neuralgias, headaches, attacks of migraine, etc.

The nutritional condition of the patient corresponds with the state of the gastro-intestinal tract and of the nervous system. Most of the patients suffering with this condition are thin or emaciated, and many become progressively more so with its persistence. Sometimes, however, typical and progressive mucous colitis may occur in neurasthenic or hysterical subjects,

who show no loss of weight at all commensurate with the gastric and intestinal disturbance.

The patient presents an appearance of anæmia, which, however, is frequently deceptive, since an examination of the blood may reveal a practically normal condition.

Complications.—Various abdominal and other conditions may occur as complications. In some cases the passage of *intestinal sand* is observed. This presents itself as a reddish deposit, resembling brick-dust, or as a grayish or white powder, which consists of phosphate and oxalate of calcium with magnesium and iron. A small amount may be passed with each movement, or sometimes large quantities may be discharged from time to time.

In some cases intense soreness of the bowels and probably actual ulceration result from the separation of firm, membranous formations. In these cases the passage of the membrane may be preceded by painful paroxysms of great violence, and may be attended with the discharge of small or large quantities of blood. After the cessation of the attack, local soreness in the left side or general abdominal tenderness may persist for some time.

Various disorders of the pelvic organs occur in women suffering with this disease; among others, endometritis, menstrual irregularities, dysmenorrhœa, and neuralgic affections localized in the pelvic region. Occasionally, membranous dysmenorrhœa and membranous cystitis have been found combined with membranous colitis. Displacements of the intestines, enteroposis, may result from this disease, or, in other cases, may predispose to it.

Among the nervous conditions associated with membranous colitis are hysteria, neurasthenia, hypochondriasis, various forms of neuralgia, pulsating aorta, and profound depression of indefinite character.

Secondary mucous or membranous colitis associated with organic diseases of the bowel, such as chronic appendicitis, adhesions, diverticula, and tumors or strictures, may be indistinguishable from the primary variety. Usually, however, the organic conditions named serve to localize the symptoms and indicate the association. In all the conditions mentioned the amount of mucus formation is comparatively slight, and is suggestive of a limited area of disease. In the less-marked cases there is merely the presence of a small amount of mucus preceding, attending, or following the passage of ordinary faecal matter. In more severe grades and generally in the later stages, more extensive involvement may occasion large quantities of mucus and membrane formation.

Prognosis.—Mucous colitis is essentially a chronic disease, which tends to continue and increase, although it is not often directly fatal. The general health may be so greatly undermined and nervous vitality so impaired that the patient falls a prey to other conditions. A large proportion of cases (perhaps 50 per cent.) eventually improve to such an extent that, although the health of the individual may be permanently impaired, the actual disease no longer manifests itself. In other cases the disease persists with more or less unabated intensity up to the time of death. Under continuous treatment some improvement is usually obtained, but the chronicity of the affection is such that few patients continue the treatment long enough to obtain the full result possible.

Treatment.—The general hygiene and the diet are the most important considerations in treatment. Outdoor exercise and suitable occupation are essential. The majority of patients suffer from a sedentary life and a

lack of suitable occupation. Not rarely, forced exercise may bring about decided relief, particularly in those cases in which general nervous depression has been augmented by lack of occupation.

The diet should be ample and varied. The patient's tendency is to restrict the dietary more and more, and finally confine herself to articles that are largely absorbed and leave little residue, thus favoring increased constipation. The first effect of a more ample diet may seem to be an increased irritation, and therefore increased mucus formation; but later, the increased strength obtained from a more adequate supply of food and the direct effect of the unabsorbed residue upon the bowel itself appears to be wholesome. No general rule can be applied to all cases. Some undoubtedly improve under restriction of diet to liquids, semisolids, and predigested foods, but in general the plan comprising a bulky diet is preferable. A change of scene may contribute largely toward making conditions better, and when distinct causes for nervous depression are discoverable, these should be remedied.

It is important that the intestinal drainage should be maintained, and when constipation is marked some form of laxative is helpful. In many cases the occasional administration of castor oil relieves the patient for some time, and may have a lasting beneficial effect. In some, the administration of very small doses of castor oil several times daily, in combination with such intestinal antiseptics as salol, betanaphthol, guaiacol carbonate, and calomel in fractional doses, may be beneficial. Now and then it is advisable to administer some form of saline purgative or to use medicinal waters for the same purpose.

When the condition seems to affect the lower bowel, colonic douches with simple saline solution, or with water containing fluid extract of hamamelis, small amounts of quinine or nitrate of silver (1 to 5000), may have a beneficial effect. It is rarely desirable, however, to persist in direct colonic medication or flushing, as too long a continuance of this plan of treatment may cause a return of the symptoms, and even an aggravation of them. Some patients are helped by the gentle injection into the colon of ten or twelve ounces of olive or cotton-seed oil at bedtime, to be retained during the night.

In some cases, active treatment of the gastric digestion may prove helpful. Increasing doses of *nux vomica* in combination with bicarbonate of soda, or the use of digestives, such as pepsin or pancreatin, may be beneficial.

Local treatment of the abdomen is useful in some cases. Abdominal massage given by a skilful manipulator, external or intra-intestinal electricity, and high-frequency currents have sometimes been used with advantage. Recently, Wright and others have treated patients by vaccination with killed cultures of the *Bacillus coli*. This method, however, rests upon no certain foundation.

Surgical measures have sometimes been used with advantage. Occasionally the removal of a chronically inflamed and adherent appendix has been followed by remarkable improvement. Some obstinate cases have been treated by attaching the tip of the appendix to an abdominal opening and flushing out the bowel daily, or at stated intervals. In other cases an artificial anus has been made in the right iliac region, and has been used to flush the colon thoroughly. The seriousness of such an operation and its general objectionable features militate against its frequent employment. It has also been suggested that the ileum be anastomosed with the rectum.

CHAPTER VII.

DISEASES OF THE PERITONEUM.

By HUMPHRY DAVY ROLLESTON, M.A., M.D. (CANTAB.), F.R.C.P.

Introductory.—The peritoneum is a serous sac of extensive dimensions; in fact, when all its reflections and fossæ are taken into account, its superficial area is but slightly less than that of the skin. In the female it differs from all other serous cavities in that its potential cavity opens indirectly externally through the Fallopian tubes, but in the male it is completely shut off from the exterior.

The peritoneum has remarkable powers of absorption both by the lymphatics and bloodvessels, as is shown by the experimental observation that, in a dog or rabbit, fluid equal to 10 per cent. of the body weight of the animal can be absorbed from the peritoneal cavity in half an hour. Fluid and soluble substances are conveyed away by the bloodvessels, whilst, in addition, the lymphatics with the aid of phagocytes carry off insoluble bodies, including microorganisms. It was formerly thought that the peritoneal cavity was in direct communication with lymphatic vessels by open apertures or “stomata,” and that it was an appendage of the lymphatic system or an immense lymph space. Muscatello¹ and MacCallum,² however, have shown that the endothelial lining of the peritoneum is everywhere continuous and complete, and that there is no actual communication between its cavity and the lymphatics, through which its contents can be pumped by purely physical means, viz., the contractions of the diaphragm.

It is true that the peritoneum covering the under surface of the diaphragm differs from the rest of the peritoneum in having a number of depressions or pits, formerly described as stomata or lacunæ, opening into the lymphatic vessels, but in reality closed by a continuous layer of endothelial cells. These cells, however, by phagocytic action, play an important part in the absorption of solid particles, and Buxton³ has shown that bacteria injected into the peritoneal cavity of animals are immediately absorbed by the lymphatics of the diaphragm and reach the general circulation in a few minutes. When the endothelial layer of the peritoneum is intact, microorganisms are probably not absorbed by the bloodvessels, but damage to these delicate cells allows rapid absorption of bacteria to take place, and hence septicæmia may result. The preservation of the endothelial lining of the peritoneum is therefore of the greatest importance. Buxton⁴ believes that the only path by which absorption into the system can take place is through the lymphatics of the diaphragm.

¹ *Virchow's Archiv*, 1895, cxlii, 327.

² *Johns Hopkins Hospital Bulletin*, 1903, xiv, 105.

³ *Journal of Medical Research*, Boston, 1907, xvi, 25.

⁴ *British Medical Journal*, 1907, ii, 1421.

In health, secretion of fluid, mainly by the great omentum, into the peritoneal cavity and its absorption therefrom, is so evenly balanced that there is just enough fluid to keep the serous surfaces moist and free from friction. In morbid conditions this state of equilibrium is disturbed and the amount of fluid may become excessive—ascites. The pathological effects produced by drying of the peritoneum are not so well known; it has been shown experimentally that it diminishes the power of absorption, and in practice it is clear that it impairs the vitality of the peritoneum, since it readily splits or tears on handling. Besides rendering the opposed serous surfaces moist and so preventing friction, the small amount of fluid plays an important part in the defence of the peritoneum against infection by exerting a bactericidal action.

The lymphatic trunks draining the peritoneal cavity run up into the thorax, especially to the anterior mediastinal glands; Durham¹ has shown that after experimental introduction into the peritoneum bacteria can be recovered from these glands after an interval of only six minutes. The peritoneum covering the diaphragm and the omentum is particularly active in this process of absorption, removing not only inert bodies but microorganisms from the peritoneal cavity; the omentum is, further, a most important factor in preventing peritonitis, since it removes microorganisms from the abdominal cavity before they can exert their pathogenic effect locally. Thus, twenty-four to forty-eight hours after the injection of bacteria into the peritoneal cavity the omentum may have rendered the peritoneum perfectly sterile, although cultures taken from the omentum itself or from the glands in the anterior mediastinum show the path by which absorption has been effected. The omentum, as is well known, has a tendency to attach itself to any inflamed organ or possible source of infection, and thus to localize the peritoneal infection. The absorptive powers of the peritoneum are diminished by the backward pressure of chronic venous engorgement, probably by lymphatic obstruction, and by less vigorous peristaltic and diaphragmatic movements. In acute peritonitis with copious fibrinous and purulent exudate there is considerable interference with absorption, and these cases are, partly on this account, more favorable than those in which there is less manifest reaction. The wide superficial area and the great absorptive power of the peritoneum explain the great gravity of diffuse peritonitis, which may thus be compared with an extensive burn of the skin. Rapid absorption of virulent microorganisms in large numbers and their toxins may lead to a fatal result before there is any manifest evidence of peritonitis; this is the condition of acute peritoneal sepsis or infection.

According to Lennander² the visceral peritoneum is quite insensitive to pain, while the parietal layer is extremely sensitive. The reason why pain is produced by traction or tension on parts of the visceral peritoneum is that the traction is conveyed to the parietal peritoneum, which is especially sensitive to dragging; the pain of colic is due to distention of the bowel, bringing tension to bear on the parietal layer of the peritoneum at the back of the abdomen and thus affecting the nerves, branches of the intercostal or lumbar trunks, supplying it. The pain of gastric ulcer, especially that excited by

¹ *Journal of Pathology and Bacteriology*, Edinburgh and London, 1897, iv, 338.

² *On the Sensibility of the Abdomen*, 1903, translated by A. E. Barker.

taking food, is probably due to peristaltic contractions of the organ leading to traction on the parietal peritoneum. The deep tenderness on pressure in localized or universal peritonitis is due to inflammation involving the parietal layer, and tenderness in inflammatory conditions of the stomach, appendix, etc., depends on lymphangitis and extension of inflammation to the parietal peritoneum. The absolute insensibility of the visceral peritoneum and abdominal viscera is seen in abdominal operations under cocaine. Further, when inflamed, the visceral peritoneum is as insensitive as in health. Kast and Meltzer,¹ from experiments on animals, confirm these conclusions for cocainized animals, but find that in animals not under the influence of cocaine the sense of pain is present in normal organs and considerably increased in inflamed organs; they therefore suggest that the insensibility observed by Lennander was due to the use of cocaine.

Comparison of the Peritoneum with Other Serous Membranes.—Like the other serous membranes the peritoneum is extremely resistant to the spread of inflammation from the tissues of the body wall covering it. It, however, is often affected, together with the adjacent pleura and pericardium, and infection and inflammation readily spread through the diaphragm. This associated inflammation is spoken of as polyorrhymenitis, or multiple serositis, and may be acute or chronic, but is more often localized or at least partially circumscribed than universal.

In common with the other serous membranes the peritoneum suffers mainly from infections derived from the organs which it surrounds and protects. Thus, like the lungs and synovial membranes, it is often affected by tuberculosis, because that form of infection commonly attacks the intestines. While unlike the pericardium, it may be regarded as immune to acute rheumatic inflammation, which is not recognized as attacking any of the organs covered by the peritoneum, with the possible exception of the vermiform appendix. Like the other serous membranes, it may be infected by the blood stream, but this hæmatogenous primary method of infection is subordinate in importance to the local spread of infection in the large serous membranes. In common with other serous membranes inflammation of the peritoneum has a great tendency to lead to adhesions.

Immunity.—Like other organs and tissues, the natural resistance of the peritoneum varies in individuals. Different parts of the peritoneal cavity probably differ in their power of resistance; the pelvic peritoneum is thicker in woman than in man, and it is possible that this has some influence in the comparative immunity of the pelvic peritoneum to infection. An attack of peritonitis leaves thickening of the affected area, even in the absence of adhesions, and thus may to some extent account for the increased resistance to infection which a past attack confers.

The defensive powers of the peritoneum are of great importance and consist in: (1) The great absorptive power of the peritoneum by means of which invading organisms are carried away before they can multiply in situ, produce toxins, and so set up inflammation. It should, however, be noted that this absorptive power may lead to a general infection or toxæmia. (2) The phagocytic action exerted by endothelial cells, polymorphonuclear and other leukocytes; the endothelial cells are always at hand, and phagocytes are especially provided by active proliferation of the cells covering the

¹ *New York Medical Record*, 1906, lxx, 1017.

omentum. (3) The bactericidal and antitoxic power of the peritoneal fluid. When occasion arises larger quantities of fluid are poured out.

It has been shown experimentally that injection of sterile fluids, nucleic acid, serum, and so forth, into the peritoneal cavity produces artificial immunity. This method has been advocated in order to prevent operative infection in man. The protection is generally believed to be due to the resulting accumulation of phagocytes in the peritoneal cavity. Buxton and Tracy,¹ however, believe that the protection resulting from intraperitoneal injection of nuclein depends mainly on blocking of the lymphatics by the inflammatory products, and that in this way systemic infection by bacteria is prevented. It has been suggested that in the early stages of peritonitis nature makes an attempt to secure protection by phagocytosis. Dudgeon and Sargent² find that a white staphylococcus, probably identical with the *Staphylococcus epidermidis albus*, described by Welch, an organism of low virulence, is usually the first bacterium to be found in the peritoneal cavity, and that it calls forth innumerable numbers of phagocytes before more virulent microorganisms gain entrance.

ASCITES.

(ἡ ἀσцитῆς ὕδρωψ = the abdominal dropsy from ἀσζή = the belly.)

Ascites is the presence of free fluid in the peritoneal cavity, and is the counterpart of a pleural effusion. The name hydroperitoneum, sometimes used as synonymous with ascites, indicates that the fluid effusion is not manifestly due to inflammation. Chronic peritonitis, however, plays such an important part in the causation of most cases of ascites, that the word hydroperitoneum is mainly employed, with the idea of excluding effusions due to subacute inflammation.

The presence of fluid in excessive amounts in the peritoneal cavity may depend on the passage of fluid in increased quantities into the sac, the rate of absorption failing to keep pace with this; or on interference with the normal process of absorption, the secretion of fluid into the cavity remaining normal or being increased. The passage of increased amounts of fluid into the peritoneal cavity mainly depends on changes in the endothelial cells of the peritoneum brought about by the action of poisons, this process occurs in inflammation, and is then spoken of as an exudation. The poisons may mainly increase the secretory activity of the cells of the peritoneum or exert a lymphagogue action. When the action of poisons cannot be assumed and the ascites appears to be the result of increased venous or lymphatic pressure—passive or mechanical effusion—the process is spoken of as a transudation. It is obvious that these two processes overlap; thus, passive venous engorgement, by interfering with the vitality of the endothelium, will produce changes analogous to those wrought by poisons.

Etiology.—The causes of ascites in the order of their importance are:

1. *Chronic Peritonitis*.—(a) Chronic peritonitis, simple, either universal or diffuse, which does not involve the whole of the peritoneum, but is more or less limited to part of the abdominal cavity, especially the upper half over the liver and spleen. (b) Associated with infection of the peritoneum

¹ *Journal of Medical Research*, Boston, 1907, xviii, 1.

² *Lancet*, 1905, i, 618.

by malignant disease. It may occur in association with the rare and interesting condition called cirrhosis of the stomach, which is now generally regarded as carcinomatous. (c) In rare instances innocent tumors, such as ovarian cystadenomas, and uterine fibromyomas, set up sufficient chronic peritonitis to cause ascites. Papillomatous disease of the ovaries may give rise to multiple implantation growths on the surface of the peritoneum, which, although not malignant, grow independently of the main tumor, irritate the peritoneum, and so cause recurrent ascites. In a case recorded by Pye-Smith,¹ the patient was tapped 299 times in nine years, sometimes twice a week, and, according to J. H. Bryant,² she occasionally relieved herself by performing paracentesis with a knitting needle. (d) Tuberculous peritonitis. Simple or "idiopathic" ascites, especially in young girls, is in many cases tuberculous.

2. *Backward Pressure*.—Backward pressure from persistent tricuspid regurgitation, and chronic venous engorgement of the liver (nutmeg liver), may give rise to copious ascites. In 100 consecutive cases in which ascites to the extent of a pint or more was found after death at St. George's Hospital, 37 were due to various forms of heart disease, mitral stenosis being present in about one-half these cases. Although in many of such instances chronic peritonitis may be the determining cause of ascites, this is not always so. In some cases of mitral disease the brunt of the backward pressure seems to fall on the hepatic veins, with the result that there is ascites without oedema of the feet. This condition, which has been called hepatic asystole, may imitate cirrhosis of the liver. In some cases of adherent pericardium there is a high grade of nutmeg liver, ascites, and little chronic peritonitis. The adherent pericardium may be the result of rheumatic fever, or be tuberculous; in some cases the tuberculous nature is only revealed by microscopic examination. Pick³ described pericarditic pseudocirrhosis, but doubt has been cast on its existence as a distinct morbid entity; it is advisable to keep an open mind on this difficult question, but probably some of the cases so described may be included under the heading either of advanced nutmeg liver or of chronic peritonitis associated with adherent pericardium. Acute cardiac failure gives rise to a small amount only of ascitic effusion, which in itself is of no clinical importance. Stricture of the hepatic veins, or thrombosis of the inferior vena cava occluding the orifices of the hepatic veins, may give rise to marked ascites.

3. *Cirrhosis of the Liver*.—Of the two forms of cirrhosis, the hypertrophic biliary or Hanot's cirrhosis, which is comparatively rare, is either not complicated by ascites or only at a very late stage, and then to a slight degree; when ascites does occur, it is probably explained by the development of multilobular (or portal) cirrhosis in addition to the existing monolobular (or biliary) cirrhosis. In portal or common cirrhosis, ascites is frequent, and is met with in 80 per cent. of the cases fatal from the effects of the disease. It may be due to the cirrhosis alone, and is then a late or terminal phenomenon, and rarely requires tapping more than twice. The causation of ascites in uncomplicated cirrhosis has given rise to some discussion. The general opinion is that it is mechanical and due to increased pressure in the portal

¹ *Transactions of the Pathological Society of London*, 1893, xliv, 111.

² *Guy's Hospital Gazette*, 1900, xiv, 148.

³ *Ztschr. f. klin. Med.*, Berlin, 1896, xxix, 385.

vein; it has usually been considered that the increased portal pressure is due to pressure on the intrahepatic branches of the portal vein, but Herrick¹ shows reason to believe that it is the outcome of a far freer communication between the hepatic artery and portal vein radicles, and that the arterial pressure is communicated to the portal vein; on the other hand, it has been argued, especially by Hale White, that this explanation is not valid and that the process is toxic and due to the presence of lymphagogue poisons in the blood, the result of failure of the antitoxic or protective function of the liver. It is not improbable that the two factors, increased portal pressure and toxæmia, work together, the first providing the vascular conditions favorable to peritoneal effusion, and the toxins damaging the endothelial cells of the peritoneum. Ascites may be merely associated with the presence of a cirrhotic liver and be caused by chronic simple or tuberculous peritonitis, or by other factors, for example cardiac failure. In these cases numerous tapings may be necessary.

4. *Other Morbid Conditions of the Liver.*—Syphilitic disease of the liver is often associated with chronic perihepatitis and peritonitis; but as it cannot always be proven that the accompanying ascites is set up in this indirect manner, syphilitic disease of the liver may be regarded as a cause of ascites. Lardaceous disease of the liver in rare instances appears to be responsible for ascites. In malignant disease of the liver ascites is common; in most cases the growths project from the capsule, irritate the peritoneum, and so induce ascites; but in some instances growths may press upon or invade and block up large branches of the portal vein inside the liver; this is especially likely to occur in the form of primary malignant disease of the liver called carcinoma with cirrhosis. In some cases new-growth may block up the capillaries in the liver so widely as to produce marked portal obstruction and ascites, as is well shown in secondary melanotic sarcoma of the organ (Hektoen and Herrick²). Ascites may occur in cases of hepatic and abdominal lymphadenoma, and is explained in the same ways as in malignant disease. From an analysis of 100 cases of ascites in Egypt, Phillips³ finds that 30 and possibly 52 per cent. of these cases were due to the effects of chronic malaria on the liver and spleen.

Perihepatitis is considered under Simple Chronic Peritonitis.

5. *Thrombosis of the Portal Vein.*—Thrombosis of the portal vein is a rare event, but usually gives rise to a rapid and extensive ascitic effusion, which speedily recurs after tapping. Pressure on the portal vein may, but does not necessarily, produce ascites.

6. *Renal Dropsy.*—In universal renal dropsy the peritoneum, in common with the pleuræ, may contain free fluid.

7. *Inflammation of Peritoneum.*—Subacute and acute inflammation of the peritoneum will, of course, give rise to an effusion.

In addition, rupture of hydatid, ovarian, or other cysts may give rise to free fluid in the peritoneal cavity; in the case of ruptured hydatid cysts, absorption will, as a rule, rapidly lead to its removal. Rupture of lymphatic vessels or lacteals will give rise to a chylous effusion into the abdominal cavity. Effusions of bile (choleperitoneum) and of pure blood (hæmo-peritoneum) are referred to elsewhere.

¹ *Journal of Experimental Medicine*, New York, 1907, ix, 93.

² *American Journal of the Medical Sciences*, Philadelphia, 1898, exvi, 255.

³ *Records of the Egyptian Government School of Medicine*, 1904, ii, 47.

Pathology.—*The morbid anatomy* of ascites is, except for the mechanical effects due to abdominal distention, that of the underlying cause, such as chronic peritonitis, cirrhosis, and new-growth.

The skin becomes stretched, and from rupture of the deeper layers of the dermis, lineæ albicantes appear; interference with the return of blood through the inferior vena cava opens up the collateral circulation in the abdominal walls between the epigastric veins below and the termination of the internal mammary and long thoracic veins above; while portal obstruction, when present, leads to the development of anastomotic channels between the systemic veins and those of the portal system. The veins of the peritoneum, especially at the back of the abdomen, become manifestly enlarged, veins in any adhesions that may be present become prominent, and the parumbilical vein in the falciform ligament may reach a large size; the veins at the lower ends of the œsophagus and rectum also become tortuous and dilated. The abdominal muscles become stretched and atrophied; the diaphragm is pushed up, and may show hypertrophy from increased work. As the result of upward displacement of the diaphragm, the lower lobes of the lungs may be collapsed. In encysted ascites the presence of adhesions limits the extent of the effusion, and may produce a loculated condition.

The characters of ascitic fluid vary according to the causes at work; thus, when there is inflammation of the peritoneum (exudates) the specific gravity and amount of contained protein are higher than in ascites associated with backward pressure (transudates). Serous ascitic fluid is clear, transparent, and greenish or faintly yellow in color. The reaction is alkaline, and the specific gravity from 1010 in mechanical effusions, or hydroperitoneum, to 1018 or higher in the exudations in subacute or chronic peritonitis.

The amounts of solids and contained proteins vary. In simple effusions due to renal disease there may be only 0.3 per cent. or less of albumin, while in inflammation the amount may be 4 per cent. In some instances, especially of ascites associated with rupture of ovarian cystadenomas, the fluid is remarkably sticky from the presence of pseudomucin. Occasionally crystals of cholesterol are present in the effusion. It may contain urea in traces or in larger amounts, and also various extractives. In cases in which repeated tapplings are necessary the character of the effusion may alter, and from added inflammatory changes in the peritoneum be of higher specific gravity and show a higher percentage of proteins.

When the patient is jaundiced the ascitic fluid contains a considerable amount of bile pigment. A blood-stained or hemorrhagic ascites may occur in the following conditions: (1) In intra-abdominal malignant disease, from leakage; this is rare in carcinoma and is more often seen in association with vascular or hemorrhagic sarcomas or endotheliomas. (2) In some cases of hepatic cirrhosis; here it is the result of traumatism, as a rule, rupture of a dilated vein in the peritoneum or in vascular adhesions, either from traction or from direct injury by the trocar. As a rule, the blood-stained character of the effusion is seen not at the first, but at a subsequent tapping. (3) A blood-stained ascites may, like a similar pleural effusion, occur in association with tuberculous infection. Red blood corpuscles have been found on microscopic examination of the ascitic fluid in tuberculous peritonitis (Grenet and Vitry¹). (4) A certain amount of blood-stained fluid, but

¹ *Compt. rend. Soc. biol.*, Paris, 1903, iv, 959.

rarely sufficient to be recognized clinically, may occur in association with strangulated hernia, volvulus, infarction of the intestines, and acute hemorrhagic pancreatitis. Sometimes the ascitic fluid clots spontaneously on standing.

The cells seen on microscopic examination vary in the different kinds of ascites. There may be endothelial cells, polymorphonuclear leukocytes, or lymphocytes, and each kind of cell may in different cases be present in greatly preponderating numbers or almost to the exclusion of the others. Examination of the cells is referred to below in the section on diagnosis, but it may be stated here that evidence is accumulating to show that, as in the cytodiagnosis of pleural effusions, a preponderance of any one form of cell is correlated with a special causal factor. Thus in ascites due to purely mechanical causes, *e. g.*, heart disease, endothelial cells are those found; in tuberculous peritonitis small lymphocytes are present in a high percentage; and in other infections the predominant form of cell is the polymorphonuclear leukocyte. In ascites associated with new-growth in the peritoneal cavity the cells of the growth or cells showing atypical mitoses may sometimes be seen; but in other cases the ascites is due to mechanical pressure exerted by the tumor, and the cells present are endothelial, as in other passive peritoneal effusions, while in other cases of intra-abdominal malignant disease subacute or acute localized inflammation may supervene and give rise to a preponderance of polymorphonuclear leukocytes.

As ascites may be due to more than one factor, for example to hepatic cirrhosis and tuberculosis combined, or to subacute inflammation supervening on a passive effusion, the cells in the fluid may show a combination of the counts characteristic of the two factors present. Thus, in hepatic cirrhosis with secondary tuberculous peritonitis there may be an approximately equal number of endothelial cells and of lymphocytes.

Of the *bacteriology* of ascitic fluid there is not much to be said. The colon bacillus and the tubercle bacillus are sometimes present. Jousset,¹ by the method of inoscopy, which consists in examining the clot produced spontaneously or by the addition of salted plasma, has proven that the tubercle bacillus is present in many cases of ascites in which a tuberculous origin was not suspected. Schaudinn and Leyden described amœboid organisms, *Leydenia gemmipara*, in ascites associated with malignant disease of the peritoneum, but their presence and nature still remain doubtful.

Chylous, Chyliform, and Milky Non-fatty Ascites.—These three forms are alike in their striking naked-eye resemblance to milk. The three forms are: (1) The true chylous ascites due to the escape of chyle into the peritoneal cavity. (2) Chyliform or fatty ascites, first recognized by Quincke, resembles in its appearances chylous ascites, but differs from it both in its causation and microscopic characters. These two forms have been much confused in the past; and as it may not always be possible to decide to which group a given case belongs, a mixed class has been described in which the fluid possesses the character of both categories. (3) The milky non-fatty ascites.

1. *Chylous ascites*, due to the escape of chyle from the lymphatics or receptaculum chyli, is a rather uncommon condition, but hardly so rare as might be gathered from Batty Shaw's collection² of only 68 reported

¹ *Arch. de méd. expér. et d'anat. path.*, Paris, 1903, xv, 289.

² *Journal of Pathology and Bacteriology*, Edinburgh and London, 1900, vi, 339.

cases in 1900. The specific gravity is usually about 1015, but considerable variations, between 1007 and 1040, have been observed, and no doubt depend on accidental factors. The fluid resists putrefaction and does not clot on standing, thus differing from chylous urine. It, however, separates into layers on standing, the fat accumulating on the surface. It is usually devoid of smell, but the distinctive odor of some special article of diet such as butter may be perceptible in it; and it has been suggested that by giving the patient some form of fat that can be readily recognized, the existence of a leak in the lacteals or thoracic duct can be determined on examination of the effusion (Straus' sign). The amount of solids is about 4 to 6 per cent.; the percentages of fat and of protein vary considerably, and may be modified by changes in the diet; there is usually about 1 per cent. of fat in a very fine state of division; the amount of protein is usually about 3 per cent. Sugar is nearly always present, but its absence does not disprove the chylous nature of the fluid. The reaction is alkaline or occasionally neutral. The important characters of true chylous ascites are (1) the finely divided condition of the contained fat, and (2) the absence of leukocytes and other cells showing fatty change.

The cause of chylous ascites is obstruction in the course of the thoracic duct or lymphatic vessels, leading to escape of the chyle inside the abdomen. Pressure from without by tumors or dense adhesions may obstruct the thoracic duct in the thorax and lead to rupture of the receptaculum chyli or to leakage from its tributaries. Thrombosis inside or infiltration of the walls of the thoracic duct or receptaculum chyli with new-growth, or even with tuberculosis, have also been recorded as responsible for chylous ascites. But in 13 cases of secondary malignant disease of the thoracic duct collected by Nattan-Larrier¹ there was no case of true chylous ascites. Intra-abdominal malignant disease, by compressing the lacteals and so raising the pressure that rupture occurs, is also a factor to be considered, but it must be remembered that malignant disease of the peritoneum is prone to give rise to fatty ascites, and that confusion between these two forms of milky ascites is not uncommon. Traumatic rupture and rupture attributed to violent coughing in whooping-cough (Wilhelm), to vomiting, and to muscular exertion have, in rare cases, been recorded. Chylous ascites has been reported as a result of rupture of a chylous mesenteric cyst. It has been met with in only a few cases of filariasis. It should be pointed out that the free lymphatic anastomosis often obviates chylous ascites when from the presence of tumors, etc., producing obstruction, it might naturally be expected. A chylous peritoneal effusion may be accompanied by a similar condition in one of the pleuræ.

2. *Chyliform or fatty* (adipose) ascites, which resembles the true chylous ascites in many respects, differs from it (*a*) in that it is due not to the leakage of chyle into the peritoneal cavity, but to the formation of fat in the peritoneal effusion, (*b*) that the globules of fat are of a much larger size, and (*c*) that cells containing fat are present in the fluid. The opalescence is not, however, always due to suspended fat, for in some instances it persists after all the fat has been removed; there is then a mixture of the two forms of adipose and milky non-fatty ascites.

Chyliform ascites is usually associated with intra-abdominal new-growth

¹ *Clinique médicale de l'Hôtel-Dieu*, Paris, 1906, v, 131.

or with chronic peritonitis, tuberculous or simple. The fat may be derived from the cells of the new-growth, or a poison generated in the growth may lead to the production of fat in the leukocytes and other cells suspended in the ascitic fluid. In cases of chronic peritonitis, the fat is thought to be formed in the cells suspended in the exudation. In some instances the fat in ascitic fluid may depend on lipæmia or the presence of fat in the blood serum, which in its turn may be the result of milk diet. It has been suggested that a lipæmic ascitic effusion may depend on disease of the pancreas (Gaultier¹). In Batty Shaw's collection of 115 reported cases of milky ascites, 27 were chyliform, but it is by no means rare and is much more common than true chylous ascites.

3. *Lactescent, Milky, Non-fatty Ascites*.—Cases have comparatively recently been described in which the milky opacity is due not to fat, as it is either absent or present in quantities insufficient to account for the milkiness, but to some other body, such as a protein resembling casein, a mucoid or nucleo-albumin, globulin, or lecithin. It has also been suggested that it depends on a limpid, non-fatty condition of the blood serum, which is particularly prone to occur in association with tubal disease of the kidneys. Although resembling chylous and chyliform ascitic fluid to the naked eye in most respects, it does not separate into layers on standing. This form of ascites may occur in cases of intra-abdominal malignant disease, but may be found in other conditions. In some instances only the later tapplings may show this condition, the earlier ones being clear and serous; or later tapplings may be more milky than the first. In such cases it would appear to be in some way related to a low form of chronic peritoneal infection and inflammation.

Physical Signs.—The abdomen is enlarged, especially in cases of chronic ascites in which the abdominal muscles have become stretched and atrophied. Measurements of fifty inches are sometimes seen. An extreme grade of abdominal distention was recorded in a woman who had been very frequently tapped and measured eleven feet around the abdomen, which contained forty-seven and a half gallons of fluid after death (Duncan).² The abdominal distention is not exclusively due to fluid, but may vary from day to day from flatulency; considerable flatulent distention often precedes the discovery of recognizable ascites. Fetal ascites may be so extreme as to obstruct the birth of the child; in a six months' fetus the abdomen has been known to contain a quart of fluid (Brodhead³).

When ascites has appeared recently the distention is mainly antero-posterior, the abdomen being prominent, especially above the umbilicus, tense, and hard. This is probably due to contraction of the abdominal muscles which are irritated by the distention. In ascites of some duration the abdominal muscles become atrophied, flaccid, and thus separation of the recti abdominales and bulging in the flanks result. It should be noted, however, that when ascites comes on in a patient whose abdominal walls are already thin and weak, as in women after repeated pregnancies, the abdomen bulges in the flanks from the outset. The distention is generally speaking uniform. But in encysted ascites there is a more or less localized prominence which does not alter in position on movement.

¹ *Compt. rend. et mém. Soc. biol.*, Paris, 1906, lxi, 429.

² *British Medical Journal*, 1906, i, 1157.

³ *American Journal of Obstetrics*, 1904, 1, 41.

It has been shown that there is a positive intra-abdominal pressure in ascites, and that this diminishes during paracentesis. Pitres¹ estimated the pressure to vary between 6 and 30 mm. of mercury, the average being 12 mm. Hg.; Quirin² found pressures of 14 to 28 mm. of mercury at the commencement of tapping, which fell 10 mm. by the end of the operation.

The skin is tightly stretched and may be shiny and present distended veins; in cases of portal obstruction, as in cirrhosis, the large veins are chiefly above and around the umbilicus and in the line of the falciform ligament; but when the obstruction is in the inferior vena cava the veins running from the middle of Poupart's ligament to the costal arch are prominent, and, when considerably enlarged and tortuous, produce the condition spoken of as a "caput Medusæ." These veins due to obstruction of the inferior vena cava are sometimes due to the pressure of enlarged glands, as in tuberculous or malignant peritonitis, but, as a rule, they are due to the pressure exerted by the ascitic fluid on the inferior vena cava, and therefore disappear after tapping. In cirrhosis both sets of veins may be present, and after paracentesis the "caval" veins disappear while the portal persist (Gilbert and Villaret³). The caval veins are most prominent in the lower and lateral part of the abdomen, while the portal anastomotic channels are most developed above the umbilicus. Occasionally a bruit is audible over these dilated veins in the epigastrium. The umbilicus becomes flush with the surface, after a time everted and tense, appearing as a thin-walled bulla, and has been known to burst in cases in which tapping has been unduly delayed. Any existing hernial sacs become distended with fluid. From stretching, the deeper layers of the skin rupture, and in a recent state reddish lines are seen, especially when the abdominal wall is lax after tapping; subsequently these lines appear as crinkled cicatrices, the well-known "lineæ albicantes." They are seen in the lower half of the abdomen. It should be remembered, however, that lineæ albicantes may be trophic and occur in persons who have gone through severe illness, or lost much of their former fat, and are not necessarily due to abdominal distention or pregnancy. In some cases œdema of the abdominal walls may accompany and obscure the signs of ascites. The costal margins are expanded and pushed out, and the abdomen moves little on respiration.

On palpation the resistance is fairly uniform. The characteristic thrill from side to side is readily brought out by flicking the surface in one flank, when a distinct impulse is felt in the corresponding position on the opposite side; in order to insure that the impulse is transmitted through free fluid inside the abdomen, and not through the abdominal parietes, an assistant's hand or a piece of cardboard should be placed vertically in the middle line. When the hand is placed flat on the abdomen and the tips of the fingers are sharply flexed, fluid between the abdominal wall and the liver or spleen, if enlarged, or an abdominal tumor, may be displaced and the firm surface of the previously obscured organ is distinctly felt; this is spoken of as "dipping."

Percussion brings out a dull note over the fluid, which collects first in the pelvis and then passes upward into the loins and hypogastric region, producing a horseshoe-shaped area of dullness. The umbilical and epigastric regions remain resonant except in those cases of chronic peritonitis

¹ *Compt. rend. Soc. biol.*, Paris, 1899, li, 674.

² *Deut. Arch. f. klin. Med.*, 1901, lxxi, 79.

³ *Rev. de méd.*, Paris, 1907, xxvii, 334.

in which, from shortening of the mesentery, the intestines cannot reach the front of the abdomen. The characteristic horseshoe-shaped distribution of dullness is sometimes disturbed by flatulent distention of the large intestine, especially in the right iliac fossa; while, conversely, dullness in the flanks may be due to liquid fæces and not to free fluid in the peritoneum. Light percussion gives a duller note than more forcible percussion, as pressure displaces a layer of fluid between the abdominal wall and an adjacent coil of distended intestine.

A very important sign of free fluid in the abdominal cavity is shifting dullness. The presence of adhesions may, of course, interfere with this test, and for its success some of the intestines must contain gas. In cases in which the amount of fluid is not sufficient to give this sign the patient may be put in the knee-elbow position and the anterior surface of the abdomen then percussed in the most dependent position for an impaired note.

Results.—Distention of the abdomen gives rise to fulness, weight, and pain in the tense parietes; “pain is a very common symptom in ascites, but is seldom or never described in accounts of that condition” (Gee). The abdominal viscera are embarrassed, so that dyspepsia and constipation are common, the latter being partly due to the flabby condition of the stretched abdominal muscles. In women the uterus may be displaced and fluctuation can be detected by the finger on pelvic examination. The abdominal muscles become atrophied, and the recti separated so that a hernial protrusion may occur. Pressure on the inferior vena cava may induce œdema of the legs, while compression of the renal veins leads to chronic venous engorgement of the kidneys and scanty high-colored urine of increased specific gravity with sometimes a trace of albumin—resembling that of the backward pressure of a failing heart. Albuminuria may, of course, be due to renal disease, such as lardaceous change, or to chronic interstitial nephritis, which is often associated with chronic peritonitis. The upward displacement of the diaphragm pushes the heart up, so that the apex beat may be in the third interspace; in some cases there is a soft, systolic apex murmur, which may disappear when the abdominal distention is relieved. The breathing may be embarrassed and cyanosis appear, the underlying condition being collapse and œdema of the lower lobes of the lungs. In rare cases the pulmonary engorgement is so extreme that there is hæmoptysis. The percussion note at the bases of the lungs behind is impaired, and Ewart² has described bilateral symmetrical triangles of dullness with a broad base corresponding with the level of the twelfth rib on each side of the vertebral column.

Diagnosis.—**Differential Diagnosis.**—The physical signs of free fluid in the abdominal cavity may, to a certain extent, be reproduced by large cysts occupying almost the whole of the abdominal cavity. In women confusion sometimes arises between a large ovarian cystadenoma or parovarian cyst and ascites. An ovarian cyst may, as shown by the history, have been first noticed in one position and subsequently spread upward into the abdomen; the maximum abdominal girth is below instead of above the umbilicus; the umbilicus may be displaced toward the thorax or to one

¹ *Clinical Aphorisms from Dr. Gee's Wards, St. Bartholomew's Hospital Reports*, 1896, xxxii, 44. Aphorism No. 128.

² *Lancet*, 1907, ii, 49.

side of the middle line, dullness is present in the middle line and not in the flanks, and there is little constitutional disturbance, the ill effects being almost entirely mechanical. On the other hand, as pointed out already, in ascites there is sometimes resonance in the flanks due to flatulence. Further, ovarian cysts may be accompanied by ascites set up by chronic inflammation of the peritoneum covering them, or they may rupture. In rare instances a huge hepatic abscess, large hydatid cyst, pancreatic cyst, or hydronephrosis has been regarded as ascites; but in these cases there may be a history of localization of the cyst at an earlier period, and even at a later stage the abdomen is hardly ever so fully occupied as to give rise to all the physical signs of ascites. In encysted peritonitis the physical signs are those of a large cyst, and the dullness does not shift in the characteristic way that it does in ascites.

Among solid or semisolid tumors the rare but remarkable fatty tumors may very closely simulate ascites and their true nature be only suspected after repeated fruitless tapplings.

Diffuse colloid carcinoma is rare, but, unless complicated by ascites, thrill and fluctuation are absent. In exceptional instances a distended urinary bladder, or even gall-bladder, has been regarded as ascites. As the patient should always pass water, or, if he cannot, be catheterized before the abdomen is tapped, the possible error of regarding a greatly dilated urinary bladder as ascites would be corrected. A greatly dilated stomach and a pregnant uterus with hydramnios have also been thought to be ascitic effusions. The absence of the concomitant signs of pregnancy should prevent ascites from being regarded as that condition; but a case of a uterine fibroid combined with ascites has been regarded as pregnancy (Montgomery¹). Great obesity with abdominal distention in women may imitate ascites, and in such cases it may be very difficult to exclude the presence of, at any rate, some free fluid in the abdomen. The distention due to tympanites should not be mistaken for ascites, since the abdomen is everywhere resonant, but when the abdominal parietes are laden with fat the percussion note may thereby be much impaired. Occasionally in adults with chronic intestinal obstruction distention of the intestines with fluid may simulate ascites, and in children with chronic enteritis this condition of pseudo-ascites has led to laparotomy. In such cases succussion due to the presence of gas as well as fluid feces in the intestines can often be obtained. Conversely, the writer has seen a case of ascites with vomiting due to gastritis and constipation regarded as intestinal obstruction.

The Diagnosis of the Cause of Ascites.—The presence of jaundice points to malignant disease or cirrhosis of the liver, deep jaundice being met with in malignant disease and comparatively slight icterus in cirrhosis. Recurrent ascites necessitating numerous tapplings indicates chronic simple peritonitis. The evidence of a growth elsewhere makes the diagnosis of malignant disease almost certain. Heart disease or tuberculosis elsewhere renders backward pressure or tuberculous peritonitis highly probable. A history or evidence of alcoholism points to cirrhosis. Thrombosis of the portal vein, which is often a complication of cirrhosis, can hardly be diagnosed, but melæna and rapid onset of ascites may suggest it. A history or the manifestations of syphilis point to this form of liver affection.

¹ *American Gynecology*, 1902, i, 449.

Enlargement of the liver, if quite smooth, may point to lardaceous disease, if rough, to cirrhosis; and if extreme or definitely nodular, to malignant disease. Less marked enlargement and irregularity may suggest syphilitic disease of the liver, a conclusion which would be strengthened by evidence of syphilis elsewhere, and may be clinched by recovery under antisymphilitic treatment. If ascites be associated with general œdema and albuminuria, renal disease is the probable cause. Multiple palpable tumors in the abdomen are in favor of malignant disease, or to a less degree of tuberculous peritonitis. Fever and concomitant pleurisy are in favor of tuberculosis, as are the scars of tuberculous glands in the neck. But enlarged glands above the left clavicle point to malignant disease.

Age has some bearing on the diagnosis. Ascites in stillborn children is generally due to inherited syphilis. Tuberculous peritonitis is the commonest cause of ascites in children and young women, and cirrhosis, simple chronic peritonitis, and malignant disease in adults over forty years of age. Examination of the fluid drawn off by paracentesis may be of practical value. If blood-stained at the first tapping, malignant disease should be suspected; microscopic examination may show pieces of malignant growth or fragments of papillomatous ovarian growths. In some cases of ascites associated with malignant disease a large number of cells showing atypical mitoses may be found, and a clue to diagnosis may thus be obtained; but in many instances the ascites is mechanical and due to pressure exerted by the growth, and shows a predominance of endothelial cells, the condition found in passive effusions, as in heart disease or cirrhosis. In other cases, again, there may be a considerable number of polymorphonuclear leukocytes, probably as the result of subacute inflammation; hence a diagnosis of malignant disease cannot always be made from examination of the cells in the effusion alone. Thickened and tenacious fluid may be due to rupture of an ovarian cystadenoma, and microscopic examination of the cells may confirm this.

Turbid fluid of a high specific gravity (1020) points to inflammation. If the predominating cells are polymorphonuclear leukocytes there is subacute inflammation; but if the cells are mainly lymphocytes, tuberculous infection should be suspected and guinea-pigs should be inoculated with the ascitic fluid. It should be remembered that many cases of clear ascitic fluid are tuberculous in origin. Examination of the fæces and the detection of tubercle bacilli has established the diagnosis in previously doubtful cases of ascites (Rosenberger¹).

In hepatic cirrhosis, when uncomplicated by superadded tuberculous or other infection, the fluid is clear and shows a preponderance of endothelial cells (Cade,² Ross,³ Gilbert and Villaret⁴). If complicated by secondary infection, as may occur after tapping, there may be a large number of polymorphonuclear leukocytes. When tuberculous peritonitis supervenes the number of small lymphocytes will increase. The main diagnostic importance of a very high percentage of endothelial cells is that it excludes acute or subacute infections and tuberculosis. If the opsonic index for tubercle bacilli be low in the ascitic fluid as compared with that of the blood serum, and

¹ *American Journal of the Medical Sciences*, Philadelphia, 1907, cxxxiv, 830.

² *Arch. de méd. expér. et d'anat. path.*, Paris, 1906, xviii, 769.

³ *Transactions of the Pathological Society of London*, 1906, lvii, 361.

⁴ *Compt. rend. Soc. biol.*, Paris, 1906, lx, 820.

if the opsonic index of the blood serum rises markedly after abdominal massage, tuberculous peritonitis is indicated. Injection of the ascitic fluid into guinea-pigs should be performed in cases of suspected tuberculous peritonitis, and the tuberculin test can also be employed.

Prognosis.—This depends in the first place on the cause. Thus, ascites in malignant disease leaves no room for hope of recovery, while that of tuberculous peritonitis is often followed by permanent cure. The ascites that recurs again and again in cases with secondary ovarian papillomas implanted on the peritoneum or in some cases of solid ovarian growths (Osler¹), may be cured by removal of the primary growth; so that the prognosis of such a case depends on detection and removal of the cause.

When ascites supervenes in an emaciated patient who is the subject of hepatic cirrhosis, the prognosis is bad, and tapping is not likely to be required more than twice before death closes the scene. The association of jaundice with ascites is rare except in malignant disease and hepatic cirrhosis, and is therefore a bad prognostic. In chronic peritonitis, however, paracentesis may be necessary for a prolonged period; Rumpf's² patient was tapped 301 times in sixteen years, and cases in which the abdomen has been punctured more than a hundred times are not very rare.

The results of the three conditions, hepatic cirrhosis, of cirrhosis complicated by chronic peritonitis, and of chronic peritonitis alone, may be arranged in this order from the point of view of the severity of the prognosis. Thus, in 38 cases of uncomplicated hepatic cirrhosis the average duration of life after the first appearance of ascites was 98.6 days; in 13 cases of cirrhosis complicated with chronic peritonitis, 360 days; and in 9 cases of uncomplicated chronic peritonitis, 624 days (Ramsbottom³). In rare cases ascites spontaneously disappears, and great diminution has been noted during an attack of fever.

Treatment.—This is in the first instance to be directed to the underlying cause if this be known and amenable to cure. Thus, ascites due to syphilitic disease of the liver should be treated by antisyphilitic remedies, and the ascites of backward pressure due to heart disease by cardiac tonics. In simple chronic peritonitis and hepatic cirrhosis no drug is known to exert any direct action on the morbid process. The palliative treatment consists in attempts to diminish the passage of fluid into the peritoneal cavity and to effect its removal. A restricted intake of fluids or a "dry diet" has been recommended, and has met with some little success, but is usually very disappointing. By excluding common salt from the dietary an attempt to diminish the effusion of fluid into the peritoneal sac, due to the retained chlorides, has been made, but the results are not very striking.

The means for the removal of fluid from the abdominal cavity fall under three heads: (1) By paracentesis, (2) by diuretics, and (3) by purgatives.

1. Paracentesis abdominis is indicated when the abdominal distention gives rise to pain, for there is nothing to be gained and a good deal to be lost by postponing the procedure. When upward displacement of the diaphragm gives rise to dyspnoea, or to collapse and œdema of the lung, as shown by moist sounds at the bases behind, or in rare instances by hæmoptysis,

¹ *Lancet*, 1907, i, 1409.

² *Deut. Arch. f. klin. Med.*, Leipsic, 1895, lv, 272. (Festschrift zu F. A. von Zenker.)

³ *Medical Chronicle*, Manchester, 1906, xlv, 7.

paracentesis should be performed without delay. Another indication is considerable diminution in the amount of urine. In cases of cirrhosis, hæmatemesis or signs of incipient delirium tremens indicate the advisability of tapping an ascitic abdomen.

The abdomen is most advantageously tapped between the umbilicus and pubes in the middle line. Sometimes from adherent omentum no fluid is withdrawn in this position, and the trocar and cannula must be introduced elsewhere. It has been recommended that the middle point of a line drawn from the anterior superior spine of the ilium to the umbilicus should be chosen, and that in order to avoid wounding the cæcum or liver the left side should be selected. In rare instances fatal hemorrhage has followed puncture in this position from a wound of the deep epigastric artery (Boidin,¹ Lian²). This accident may be suspected if, on withdrawing the cannula, pure blood comes in spurts, and should be met by ligature of the wounded vessel.

Before the abdomen is tapped the urinary bladder should be emptied, and the site of the puncture percussed and be found to be dull. The patient should lie on his back with the head slightly raised; the skin in the neighborhood of the intended puncture should be cleaned and rendered aseptic. If necessary, the skin may be anesthetised by ethyl chloride. A cannula of a comparatively small size, such as that employed by the late Dr. R. Southey, Physician to St. Bartholomew's Hospital, 1868 to 1883, should be used, so as to remove the fluid slowly. Rapid removal of fluid is dangerous, as it may lead to faintness. The sterilized Southey's trocar and cannula should then be introduced, the trocar withdrawn, and the shield fixed in position by pieces of plaster. The fluid is then allowed to run away through a thin, india-rubber tube. This usually takes some eight to twelve hours to drain away, and during this period the abdomen may be protected by a cradle, and a many-tailed bandage or a binder should be kept comfortably tight over the abdomen. If the flow stop before the abdomen has been fairly well evacuated, the india-rubber tube should be "milked," starting from the cannula. When the fluid has finally ceased to run, the cannula should be withdrawn and the small wound closed by a pad of absorbent cotton-wool soaked in collodion. The abdomen should be bandaged or, as has been recently recommended, tightly strapped with adhesive plaster, in order to prevent rapid accumulation of the fluid and flatulent distention. In some instances good results have followed the injection through the cannula, after the abdomen has been nearly emptied, of a dram of adrenalin chloride, 1 to 1000, in an ounce of water. Probably adrenalin acts by retarding both transudation and absorption, and, as shown by Meltzer and Auer,³ these effects are more lasting than its transient effect on blood pressure. By injecting oxygen through the cannula after tapping, good results have been obtained in tuberculous peritonitis, but no results in the ascites of hepatic cirrhosis (Schulze⁴). In addition to wounding a bloodvessel in the abdominal wall, there are very few dangers connected with the operation of paracentesis. In extremely rare instances acute pulmonary œdema has followed paracentesis of the abdomen. Wounding of the abdominal viscera should be avoided if proper care be taken. As has been mentioned

¹ *Bull. Soc. anat.*, Paris, 1903, 415.

² *Ibid.*, 1907, 665.

³ *Transactions of the Association of American Physicians*, 1904, xviii, 207.

⁴ *Mitt. a. d. Grenzgeb. Med. u. Chir.*, Jena, 1907, xviii, 180.

elsewhere, repeated tapping may set up some degree of chronic peritonitis; acute peritonitis is, with ordinary attention to antiseptic precautions, very rare. Prolonged or permanent drainage is not advisable.

2. Diuretics are disappointing in the treatment of ascites, and their use should not be persisted in when there are indications that paracentesis is necessary. They sometimes appear to succeed after paracentesis, probably because the kidneys are then in a better position to act; for, as the result of a large ascitic effusion, pressure is exerted on the renal veins and thus a condition of chronic venous engorgement is induced. The diuretics employed are numerous, and the use of any one will to some extent be determined by the cause of the ascites. Thus, digitalis, which is useful in cardiac cases, should not be employed in renal cases with excessively high tension. In heart cases Baillie's or Addison's pill, containing digitalis, squill, and mercury, is commonly used; citrate of caffeine, diuretin, or theocin combined with digitalis may also be given. In ascites due to other causes acetate, citrate, or tartrate of potash, spirit of juniper, copaiba resin (in capsules), and urea may be tried. Apocynum has been recommended, but it has not been successful in my hands and is a gastro-intestinal irritant.

3. *Purgation*.—Moderate purgation by calomel or jalap and salts, such as tartrate of potash or sulphate of magnesium and sodium combined, may be tried; but drastic purgatives, such as elaterium and gamboge, should be avoided, as the patient's strength may be seriously impaired. Such treatment in the past has been thought to have purged patients to death.

Operative Treatment.—In tuberculous peritonitis and hepatic cirrhosis, operative treatment has been employed with a view of a more or less radical cure. In tuberculous peritonitis opening the abdomen and removing the fluid, which has a lower opsonic index than that of the blood, is followed by the appearance of peritoneal fluid with a high opsonic index, and we are thus provided with an explanation of the way in which simple laparotomy does good (White¹). On the other hand, statistics show that medical treatment may give as good results as surgical, and it is only fair to remember that the cases treated surgically are selected, while those treated medically are not.

The ascites of hepatic cirrhosis has been extensively treated by the production of artificial peritoneal adhesions, with the object of increasing the collateral venous circulation between the general and portal systems, and thus relieving the increased pressure in the portal vein. This method was independently conceived by Talma and by D. Drummond and Morison, and the operation is often spoken of as the Talma-Morison procedure. In 1906 Sinclair White² tabulated the results of 227 cases, and found that 37 per cent. were cured, 13 per cent. were improved, in 15 per cent. the operation failed to give relief, and in 33 per cent. it was followed by death.

Hæmoperitoneum.—The presence of pure blood free in the peritoneal cavity is a somewhat rare condition, and must be distinguished from blood-stained ascitic effusions. The causes of intraperitoneal hemorrhage are: (1) Rupture of ectopic gestation; this is the most frequent cause; of 20 cases of intraperitoneal hemorrhage examined by Dudgeon and Sargent,³ 17 were due to this cause. (2) Traumatic rupture of the spleen, liver, or mesentery.

¹ *Transactions of the Royal Academy of Medicine, Ireland*, 1906, xxiv, 410.

² *British Medical Journal*, 1906, ii, 1287.

³ *Lancet*, 1905, i, 474.

(3) Rupture of aneurisms of the cœliac axis, or of the superior mesenteric, hepatic, or other intra-abdominal arteries. An aneurism of the right branch of the hepatic artery inside the liver has been known to produce rupture of the capsule and intraperitoneal hemorrhage (Biekhardt and Schümann¹). Aneurisms of the abdominal aorta usually rupture behind the peritoneum in the first instance, but may subsequently leak into the peritoneal cavity. (4) From leakage of a hemorrhagic or vascular new-growth, such as an angiosarcoma of the liver or adrenals. (5) From rupture of varicose or dilated veins; thus, most profuse hemorrhage may occur from rupture of a dilated vein on the surface of a subperitoneal fibromyoma. Rupture of a varicose vein in the broad ligament is rapidly fatal, but is fortunately extremely rare. The writer has seen a large quantity of blood in the peritoneal cavity from traumatic rupture of a vein in peritoneal adhesions. (6) From rupture of a hemorrhage into the suprarenal bodies. (7) In very rare instances repeated peritoneal hemorrhages may occur without any known cause. In the remarkable case recorded by Cheeseman and Ely² the abdomen was tapped forty-three times in five years and spontaneous cure then occurred. Both pleuræ had previously presented similar recurrent hemorrhagic effusions.

Intraperitoneal hemorrhage, although sterile at first, rapidly becomes infected. In 20 cases examined by Dudgeon and Sargent, only one was completely sterile, and that was operated upon two hours after the onset; the organism isolated by these observers was a white staphylococcus. This observation explains (a) the rise of temperature which may follow intraperitoneal hemorrhage, and was formerly referred to the absorption of fibrin-ferment; and (b) the onset, if the patient survived sufficiently long, of peritonitis.

Symptoms.—There is sudden pain which marks the extravasation of blood into the peritoneal cavity. This is followed by faintness, rapid and feeble pulse, restlessness, sighing respiration, vomiting, attacks of colicky pain, bloodlessness and extreme pallor, sweating, and collapse. In hemorrhage due to a ruptured ectopic gestation marked acetoneuria has been described.

In some cases, for example, rupture of an aneurism, death occurs at once. In other cases, especially when the blood is poured out less rapidly, the clinical manifestation may imitate those of perforative peritonitis due to rupture of a gastric ulcer.

Sequels.—If the patient is not operated upon and survives, acute general peritonitis will follow, unless, indeed, the extravasation be sufficiently localized to become encysted and form a peritoneal sanguineous cyst.

Diagnosis.—In the presence of signs and symptoms of internal hemorrhage and a history of recent and severe traumatism the diagnosis is easy. On the other hand, cases of ruptured ectopic gestation may so closely resemble acute perforative peritonitis that the condition is not suspected until the abdomen is opened. When signs of collapse occur in a patient known to have an intra-abdominal tumor the occurrence of hemorrhage may be suspected.

Prognosis.—Prognosis is best in ruptured ectopic gestations, but it is necessarily fatal in ruptured aneurisms and eventually, of course, in leaking malignant growths.

¹ *Deut. Arch. f. klin. Med.*, Leipsic, 1907, xc, 288.

² *American Journal of the Medical Sciences*, Philadelphia, 1899, cxviii, 166.

Treatment.—When the cause is a ruptured extra-uterine gestation or spleen the only course is removal of the ruptured organ.

Choleperitoneum.—Rupture of the bile passages with effusion of bile into the cavity of the peritoneum usually sets up peritonitis, because the conditions responsible for the accident entail microbic infection of the bile; but when aseptic bile is poured out into the peritoneal cavity there is no inflammation; and in rare instances large bilious effusions result. This condition was named choleperitoneum by Dévè,¹ and in 1906 Beaudet² collected 47 cases due to rupture of a hydatid cyst which either before or after the rupture was in communication with a bile duct. The abdomen usually enlarges slowly, but it may do so rapidly. There is no fever, and it is remarkable that, in spite of the marked absorptive powers of the peritoneum, jaundice does not occur. The effusion has a great tendency to recur, and several tapings may be required.

ACUTE DIFFUSE PERITONITIS.

Although acute peritonitis means acute inflammation of the peritoneum, the term is used to describe cases showing the clinical effects of acute infection of the peritoneum, namely, septicæmia and toxæmia, in which there may be very little or no manifest inflammatory reaction on the part of the peritoneum. These cases are more suitably described as “acute peritoneal infection.” Thus, although it may sound paradoxical, the most severe cases of peritoneal infection often show the least evidence of inflammatory reaction. Peritonitis is indeed a salutary process in so far that it prevents excessive absorption from the peritoneum, leads to destruction of micro-organisms that have gained entrance into the cavity of the abdomen, and by the formation of fibrin and adhesions prevents the spread of infection throughout the peritoneal cavity.

Acute diffuse peritonitis is not necessarily universal and co-extensive with the whole area of the peritoneum. Strictly speaking, cases of acute peritonitis might be divided into (1) circumscribed, (2) diffuse or spreading, and (3) universal or total. But in practice acute peritonitis is usually considered as either (1) circumscribed or localized, or (2) general or diffuse.

There is some unavoidable vagueness about the terms general and diffuse as descriptive of the extent of the peritonitis seen at an operation; thus, the word employed may be meant to imply universal infection and inflammation of the peritoneum throughout the whole of the abdomen, or only that at the time of the operation the peritonitis was not strictly localized. It is therefore somewhat difficult to judge of the value of the statistical results in cases of peritonitis described as diffuse or general. It is, however, usually impossible, and certainly inadvisable, to determine during an operation whether the peritonitis is absolutely universal or only diffuse, and in any case universal peritonitis is a later stage of the diffuse form. It has, therefore, been suggested that the term universal should be employed instead of general, and progressive instead of diffuse. The term diffuse will be employed here.

¹ *Rev. de chir.*, Paris, 1902, xxvi, 67.

² *Gaz. des hôp.*, Paris, 1906, 1065.

Pathogeny.—Since chemical poisons, such as turpentine, croton oil, and nitrate of silver, when introduced into the peritoneal cavity of animals, can undoubtedly set up acute inflammation, the question arises whether general peritonitis in man is ever due to toxins in the absence of bacteria. In other words, whether chemical peritonitis in contradistinction to bacterial peritonitis occurs in man. In cases of bacterial infection of the vermiform appendix or gall-bladder local peritonitis in the immediate neighborhood may conceivably be due to diffusion of toxins through the walls of the inflamed viscera. Cultures and coverslip preparations from cases of this kind in an early stage may, indeed, show an absence of microorganisms. This inflammatory reaction would, however, be strictly localized, and in any case after a short time microorganisms would pass through the damaged walls of the affected viscera.

Sterile bile and urine do not set up peritonitis, and although trypsin may produce a hemorrhagic inflammation, the secretions of the pancreas and stomach are, when they gain entrance into the peritoneal cavity, practically always mixed with the contents of the alimentary canal, and therefore with bacteria. In cases in which corrosive poisons are taken by the mouth into the alimentary canal, the damage to the walls of the stomach and intestines would enable bacteria to pass into the peritoneum. Moreover, in inflammation of the peritoneum set up by chemical means, bacteria would almost certainly gain an entrance sooner or later from the alimentary canal or by the blood stream. It is true that cases of peritonitis occur in which cultures from the peritoneum prove sterile, but Durham¹ has shown how rapidly microorganisms are removed from the peritoneum, and that in cases of peritonitis in which the exudation is sterile the omentum contains bacteria. Whilst, therefore, the possibility of chemical (aseptic) peritonitis may be admitted, it is safe to assume that in practice all cases of acute peritonitis are due to bacterial infection.

Although acute diffuse peritonitis is from a practical point of view always due to bacterial infection of the peritoneum, the mere entry of microorganisms into the peritoneal cavity is not alone enough to produce peritonitis. The defensive powers of the peritoneum, viz., the phagocytic action of the endothelial and other cells, the bactericidal power of the peritoneal fluid depending on the presence of antibodies, and absorption—which destroy and remove the invading bacteria—are able to prevent the production of inflammation. With regard to absorption from the peritoneal cavity, it should be pointed out that, while the removal of bacteria in comparatively small quantities and their destruction in the lymphatic glands and other organs prevent the onset of peritonitis, and are therefore beneficial, the passage of large numbers of virulent bacteria into the general circulation, which occurs when the endothelial cells of the peritoneum are damaged, gives rise to a grave and often fatal bacteriæmia. Death from “shock” after perforation may in all probability be explained in this way. The absorptive powers of the peritoneum also militate against peritonitis by removing fluid in which microorganisms might multiply. The defensive powers of the peritoneum have been divided by Andrewes² into those which are physiological, or the “first line of defence,” and those which come into play when inflam-

¹ *Journal of Pathology and Bacteriology*, London and Edinburgh, 1897, iv, 357.

² *General Pathology and Bacteriology of Acute Peritonitis*. *System of Medicine* (Allbutt and Rolleston), 1907, vol. iii, 897.

mation is set up, or the "second line of defence." These two lines of defence merge into each other, for example, in phagocytosis, but the formation of fibrinous adhesions which localize the infective focus is a frankly inflammatory process.

When the defensive powers of the peritoneum are inhibited or prevented from exerting their full effects, bacterial invasion is enabled to set up peritonitis. Factors which reduce the resistance of the peritoneum, such as the sudden entrance of poisonous fluids, drying or traumatism during the course of an operation, and possibly cold, may interfere with the absorptive and bactericidal powers of the peritoneum. Again, the presence of solid bodies, such as particles of food, faeces, blood clot, concretions, or foreign bodies, or of ascitic fluid, may protect the bacteria, provide a nidus for their multiplication, inhibit absorption, and so enable them to produce their toxins and thus set up inflammation of the peritoneum.

Etiology.—Acute diffuse peritonitis may be (1) primary or (2) secondary.

1. The term primary idiopathic peritonitis has been employed in two senses. Formerly it was used to imply that inflammation attacked the peritoneum alone, the rest of the body being healthy, and that its cause and origin were unknown. Cases of peritonitis formerly thought to be due to rheumatism and to cold came under this heading. More recently, since it has been recognized that acute peritonitis is practically always infective, the term primary peritonitis has been applied by Flexner¹ to cases in which, as there is no local focus in the abdomen to account for the infection of the peritoneum, it is assumed that microorganisms have reached the peritoneum by the blood or lymph stream. Hæmatogenous peritonitis occurs as a terminal infection in chronic nephritis, arteriosclerosis, cancer, and other conditions which reduce the bactericidal power of the blood. Some cases of peritonitis complicating erysipelas or other infections, and about half of the instances of pneumococcal peritonitis, would also appear to be primary and so of hæmatogenous origin. Since a minute local focus, such as a microscopic abscess in the appendix, may be easily overlooked, a most exhaustive post-mortem examination is necessary before a case of primary idiopathic peritonitis can be accepted, and many writers are somewhat unwilling to admit its existence. Of this, however, there can be no doubt, and probably about 10 per cent. of the fatal cases of general peritonitis are of this nature. It occurred in 12 out of 106 cases examined by Flexner and in 9 out of 105 cases at the Massachusetts General Hospital (Manahan²). The infection is usually single; thus out of Flexner's 12 cases no bacteria were found in 2, in 9 there was a single infection, and in 1 a mixed infection.

2. *Secondary peritonitis*, in which infection is due to some local lesion either in or in the immediate neighborhood of the abdomen, accounts for the great majority of cases of acute diffuse peritonitis.

(a) Wound or operation introducing infection into the peritoneal cavity, *e. g.*, after herniotomy. To this group Flexner applies the term exogenous. But it must, of course, be remembered that postoperative peritonitis may be due to bacterial infection derived from the viscera.

(b) Due to infection derived from the abdominal viscera. This may depend on gross perforation of their walls or be due to inflammation allowing

¹ *Philadelphia Medical Journal*, 1902, ii, 1019.

² *Boston Medical and Surgical Journal*, 1905, clii, 345.

microörganisms to pass through the intestinal walls. This is Flexner's endogenous group. It will be most convenient to give a list of affections of the abdominal viscera which may give rise to acute peritonitis.

Stomach.—Perforation of a gastric ulcer; perforation due to rapidly growing, soft, and necrosing carcinoma; acute suppurative (or phlegmonous) gastritis; acute gastritis due to corrosive poisons.

Small Intestines.—Traumatic rupture, perforation by sharp foreign bodies; perforating ulcers, duodenal; jejunal peptic ulcer, occurring in cases of gastrojejunostomy, especially, it is said, after the anterior operation; typhoid ulcer (peritonitis in rare instances occurs in the absence of any gross perforation); uræmic ulcer; acute enteritis; strangulation, kinking, volvulus, intussusception; internal hernia; infarction.

Appendix.—Perforation as a result of acute infective¹ inflammation; from rupture of an abscess in the walls of the appendix or in its neighborhood, or from acute appendicitis as already mentioned. An abscess in the appendix so minute as to be easily overlooked may rupture into the peritoneum and set up peritonitis, which may thus appear to be primary or idiopathic.

Colon.—Perforation, traumatic or by a foreign body from within. Perforating ulcer, stercoral dysenteric; in connection with carcinoma, either from rupture of a distention ulcer, or leakage through necrosing growth. Extremely acute inflammation, in strangulation, volvulus, intussusception. Wounds, perforation of the rectum by a bougie, nozzle of an enema syringe.

Gall-bladder.—Acute, phlegmonous, or gangrenous cholecystitis. Rupture of an inflamed gall-bladder.

Liver.—Rupture or leakage of an abscess or of a suppurating hydatid cyst. Abscesses may be multiple and due to arterial pyæmia, infection of the portal vein (pylephlebitis), or of the bile ducts (suppurative cholangitis). In newborn infants phlebitis of the umbilical vein gives rise to peritonitis.

Spleen.—Rupture of an abscess, or of a suppurating infarct.

Mesenteric Glands.—Rupture of a suppurating lymphatic gland into the general cavity of the peritoneum.

Ulceration and perforation of the *urinary bladder* or of *sacculi* in connection with it.

Suppurating and gangrenous ovarian cysts.

Pyosalpinx and infection spreading from the Fallopian tubes. The importance of this factor has probably been overestimated in the past. Riedel¹ has shown reasons for believing that salpingitis is a more important cause of general peritonitis in girls under ten years of age than in adult women.

Perforation of the uterus by a sound.

Rupture of an abscess into the peritoneal cavity, especially of a peri-appendicular abscess; this may follow rough manipulation. It is remarkable how rarely erysipelas or suppuration in the abdominal parietes gives rise to peritonitis; this probably depends on the lymphatics being independent. Although peritonitis may lead to infection of the pleuræ, an empyema hardly ever ruptures into the abdominal cavity. Peritonitis readily spreads from the pancreas to the peritoneum of the lesser sac. Retroperitoneal

¹ *Arch. f. klin. Chir.*, Berlin, 1906, lxxxi, 186.

suppuration, such as that due to tuberculous ostitis of the spine or to renal calculi seldom infects the peritoneum. In infants acute peritonitis is usually due to infection from the umbilicus.

As giving the relative importance of the various starting points of acute peritonitis, Benda's¹ 446 cases of acute diffuse peritonitis examined after death may be referred to. The vermiform appendix was the starting point in 115 cases, the stomach and duodenum in 68, the rest of the intestine in 118, the female genitals in 81, the gall-bladder in 10, the kidney and urinary bladder in 10, the pancreas in 2, the spleen in 1; 35 were unknown, 4 were postoperative, and 2 were described as hæmatogenous (nephritis and rheumatic fever).

The *sex* and *age* incidence depend on those of the commonest causes of peritonitis, perforating gastric ulcer and pelvic infections being the most frequent etiological factors in women, and appendicitis in children and young males. In 200 fatal cases at St. George's Hospital there were 107 males and 93 females; the average age of the 200 cases was 32 years, being 34.26 in the males and 29.3 years in the females. The largest number of cases occur between twenty and forty years of age.

Bacteriology.—Although a great deal of work has been done on this subject our knowledge is far from complete. In the following account the results obtained by Dudgeon and Sargent will be freely utilized. A large number of microorganisms have been found in human peritonitis, the most important of which will now be considered. In many instances there is a mixed infection, and difficulty arises in apportioning the share that the organisms present have taken in producing peritonitis. The organisms found in peritonitis are in nearly all instances morphologically the same as those commonly present in the healthy intestine.

The *Bacillus coli communis* is very commonly found, and considerable discussion has taken place whether its importance is commensurate with its frequency. It has been regarded as the all-important factor in peritonitis, and, on the other hand, it has been recently thought that the *Bacillus coli*, being more hardy than other bacteria, may overgrow them and so be found postmortem, to the exclusion of other microorganisms which are the real factors in producing peritonitis. That this disappearance of the original organism can occur has been shown experimentally by Klein, and in man an organism, the pneumococcus, found in pure culture at operation, has not been obtained after the death of the patient, while *Bacillus coli* was (Charrin and Veillon²). But these observations do not in any way prove that the colon bacillus is not an important cause of peritonitis. It is naturally found most often in cases of peritonitis due to infection from the alimentary canal, and is very frequently present in association with some other organism; in 56 cases of peritonitis due to bowel infection, Flexner found the *Bacillus coli* in 43, in pure culture in 8, and in association with other organisms in 35. Dudgeon and Sargent,³ as the result of bacteriological examination of 270 cases, came to the conclusion that the colon bacillus is the most important factor, and that it is found in the largest number of fatal cases. Its virulence varies greatly, and while it is less virulent than

¹ Quoted in *A System of Surgery*, von Bergmann, von Bruns, and von Mikulicz, translation, vol. iv, 165.

² *Compt. rend. Soc. biol.*, Paris, 1893, xlv, 1057.

³ *The Bacteriology of Peritonitis*, Constable & Co., London, 1905.

the streptococci and *Bacillus pyocyaneus*, it is more frequently present. With mixed cultures a very virulent peritonitis is set up.

A white *staphylococcus*, which is not the same as the *Staphylococcus pyogenes albus* described by Rosenbach, and has often been regarded as due to contamination from the skin, and is probably Welch's *Staphylococcus epidermidis albus*, has been found by Dudgeon and Sargent to be the first organism to appear in the peritoneal cavity in cases of early peritonitis, and to pass through the walls of the inflamed intestine before more virulent organisms. They found this microorganism in 108 out of 258 cases of different peritoneal lesions.¹ It is an organism of low virulence, and, as mentioned above, gives rise to an exudation rich in phagocytes and thus exerts a protective action against more virulent microorganisms.

Streptococci are found in a considerable percentage of the cases. Flexner found them second in order of frequency both in his primary and secondary groups of peritonitis. As streptococci are among the normal saprophytes of the intestinal canal, it is natural that they should be commonly present in cases of peritonitis due to intestinal lesions, but it must be remembered that the *Streptococcus pyogenes* has not been found normally in the alimentary canal, and that streptococci found in the peritoneum are not necessarily *Streptococcus pyogenes*, and may be the saprophytes of the intestine (Andrewes and Horder²). Peritonitis due to *Streptococcus pyogenes* is the most severe and fatal form; there is little or no local reaction, the phagocytes being powerless against the virulent microorganisms which, as shown by Buxton's³ experiments on animals, are rapidly absorbed into the circulation and give rise to bacteriæmia, proving fatal in the course of 24 to 48 hours from liberation of their endotoxins.

Staphylococcus pyogenes aureus was found by Flexner in 15 out of 34 cases of exogenous peritonitis, being in pure culture in 12 and mixed in 3. It was present in 3 cases only in 39 cases of endogenous peritonitis. It gives rise to a virulent form of peritonitis.

Bacillus pyocyaneus is responsible for a certain number of cases of acute peritonitis, and ranks next to the *Streptococcus pyogenes* in the virulence of the resulting peritonitis. It does not produce green pus in the peritoneal cavity, as it often does in a local abscess. Dudgeon and Sargent have shown that *Bacillus coli* will not grow in the presence of *Bacillus pyocyaneus*.

Pneumococci have been isolated in a small percentage of cases, but much attention has been paid to pneumococcal peritonitis, as it is comparatively often primary. It has been found in cases of peritonitis due to appendicitis, or to perforation of a gastric ulcer, peptic or malignant.

Gonococcal peritonitis is infrequent, and when it does occur is chiefly found in women and children. No doubt some cases of gonococcal peritonitis are mixed infections, and the severity of such cases is due to the other microorganisms.

Bacillus typhosus has in a few instances been found in cases of peritonitis due to perforation of typhoid ulcers, and even after rupture of the spleen. But it is combined with saprophytic organisms, and it is doubtful what importance should be attached to its presence.

Other organisms, such as *Bacillus proteus*, *Bacillus anthracis*, are occa-

¹ *Lancet*, 1906, ii, 1337.

² *Ibid.*, 1906, ii, 708.

³ *Journal of Medical Research*, Boston, 1907, xvi, 39.

sionally found. With regard to anaërobic organisms, there has been a good deal of discussion; Veillon and Zuber and Tavel and Lanz conclude that anaërobic organisms are of importance in the production of appendicitis and peritonitis, whilst Dudgeon and Sargent employed strictly anaërobic precautions in a number of cases of appendicitis and in other varieties of peritonitis, but failed to find anaërobic organisms except in one case of gangrenous appendicitis with abscess in which *Bacillus aërogenes capsulatus* was found in addition to *Bacillus coli* and a streptococcus. As the result of the presence of anaërobic organisms the peritonitic exudate may become putrid.

Hill and Fisch¹ report a case of peritonitis due to the influenza bacillus, and refer to another case examined by von Recklinghausen. One case occurred in the Johns Hopkins Hospital.

It may be well to summarize the data at our disposal as to the bacteriology of peritonitis due to various anatomical lesions. In perforative peritonitis due to gastric ulcer, the pneumococcus and a streptodiplococcus, an organism of low virulence (Dudgeon and Sargent), have been thought to be the responsible organisms. In perforation of the small intestine the *Bacillus coli* and streptococci, and in appendicitis various organisms, especially streptococci (Low, Lartigau), *Bacillus coli*, and diplococci have been regarded by different observers to be the causal organisms. Krogius, A. O. J. Kelly, and Dudgeon and Sargent are opposed to the view that streptococci commonly give rise to appendicitis. In puerperal peritonitis streptococci are most often the responsible organism.

Morbid Anatomy.—The morbid appearances seen in the abdomen necessarily vary with the causes and stages of acute peritonitis. In the most rapidly fatal cases, in which death is due to septicæmia from free absorption from the peritoneal cavity, there may be little or no evidence of morbid change in the peritoneum. In other cases the earliest visible change is congestion of the small bloodvessels of the peritoneum; this is not uniformly distributed, but is naturally best marked and at first confined to the starting point of the inflammation and to the omentum which is often adherent to the focus of infection. Further it is more marked on the visceral than on the parietal layer of the peritoneum, and is in most instances not diffuse, but is confined to longitudinal lines which correspond with the spaces left between contiguous and distended coils of intestine. The next change is the exudation; at first there is slight dulling of the smooth peritoneal surface, which appears finely granular, and on scraping yields a little fibrin. This fibrin is sticky and as it increases in amount glues the intestines together and collects in the triangular chinks left between adjacent coils. At first the fibrin is thin and clear, but it becomes opaque, and may be yellow or even of a green tint. Large masses of fibrin may eventually form and show a somewhat spongy or reticulated structure. This fibrin serves a useful purpose in limiting the field of infection and tending to localize the peritonitis, and also in protecting the peritoneal endothelium and so preventing absorption of bacteria and their toxins into the blood stream in overwhelming quantities.

The fluid portion of the exudation, usually comparatively small, may in some instances be considerable (several quarts). It collects between the

¹ *St. Louis Medical Review*, 1903, xlviii, 55.

intestinal coils; during life it is often mainly in the loins, but after death it is more commonly seen in the pelvis. Its characters vary much in different cases: it may be (1) serous; (2) serofibrinous, turbid, with pieces of fibrin floating in it, and of a specific gravity of 1018 or upward; (3) fibrinous; (4) fibrinopurulent; (5) purulent; (6) sanious; (7) putrid, due to the presence of putrefactive anaërobic organisms, such as the *Bacillus capsulatus aërogenes*. In such cases there may be free gas in the peritoneal cavity without any perforation of the hollow viscera.

The character of the exudate differs in different forms of infection. In the virulent streptococcal infection there is a little thin, odorless fluid; in gonococcal infection there is a dry, fibrinous exudate with hardly any pus or serum; in pneumococcal peritonitis the exudate resembles that in a pneumococcal empyema, and in colon infections there is a thick, creamy pus.

The *progressive fibrinopurulent form of peritonitis* described by Mikulicz is transitional between circumscribed and diffuse peritonitis. There is a gradually extending area of foci of pus enclosed in fibrinous adhesions, and in rare instances the whole of the peritoneal cavity may be involved. From rupture of an encysted collection of pus general purulent peritonitis may be set up.

The intestines are in a state of paralytic distention, the walls somewhat swollen, oedematous, and softened from inflammation so that the peritoneum readily strips off and the intestine may rupture if pulled upon. In children there may be retrograde intussusceptions produced during the death agony. The peritoneum may show scattered small hemorrhages, and small extravasations of blood are not uncommon in the ovaries. The omentum is often adherent to the starting point of the peritoneal infection, *e. g.*, the appendix.

The lymphatics become filled with exudation and cells, and infection is, as shown by Durham¹ both experimentally and by observations on fatal cases of peritonitis in man, conveyed to the lymphatic glands in the anterior mediastinum; these glands are swollen, reddened, and contain microbes. A diagnosis of peritonitis is, indeed, sometimes possible from examination of these glands before the abdomen is opened. It is significant that in cases fatal from "shock" or "collapse" within twenty-four hours after laparotomy those glands are infected, showing that the cause of death is really infection.

As the result of toxic absorption the solid viscera, such as the liver, pancreas, spleen, kidney, show cloudy swelling of their cells and areas of focal necrosis. Twenty-three cases of acute or organizing peritonitis showed liver necroses in 10, and pancreatic necroses in 6 (Whipple²). In rare instances other serous membranes, especially the pleuræ, are inflamed as the result of a concomitant infection—an acute polyoromenitis.

In prolonged cases, especially in pneumococcal and puerperal peritonitis, local abscesses may form. These if left alone may perforate in various directions, into hollow viscera, or even externally at the umbilicus.

Microscopically the endothelium of the peritoneum, especially of the omentum, early shows proliferative changes; the cells enlarge; their nuclei become swollen and divide by direct and then by indirect nuclear division.

¹ *Journal of Pathology and Bacteriology*, Edinburgh and London, 1897, iv, 361.

² *Johns Hopkins Hospital Bulletin*, 1907, xviii, 391.

The cells ("macrophages" of Metchnikoff) thus produced vary in size, but some are indistinguishable from lymphocytes. The surface of the endothelial cells is covered by a layer of fibrin containing leukocytes, and there is small round-celled infiltration of the underlying (subendothelial) tissues. Coarsely granular eosinophile cells constitute a considerable proportion of the immense number of leukocytes attracted to the bloodvessels of the part; this local accumulation is correlated with the diminution of eosinophile cells in the peripheral circulation (Opie¹). At a later stage the cell infiltration of the underlying structures becomes more intense and extensive, and the peritoneal endothelium is destroyed.

If the inflammatory process be short-lived the endothelium is not destroyed, and the fibrin is eventually absorbed, so that the formation of permanent adhesions is prevented. If the inflammatory process be continued the endothelial cells are destroyed and permanent adhesions form. There is some divergence of opinion as to the method by which adhesions are formed; some have considered that they may result from endothelial proliferation and metaplasia, but it appears more probable that peritoneal adhesions are formed from proliferation of connective-tissue cells.

The exudation and fibrin formed on the surface of the peritoneum are protective by (a) enabling phagocytosis and bacteriolysis to take place, (b) by blocking up the lymph channels and so preventing absorption from the peritoneal cavity, and (c) by preventing the passage of microorganisms from the lumen through the walls of the inflamed intestine.

Cytology.—The cells normally present in peritoneal fluid have been mainly studied in animals, and the descriptions given by Kanthack and Hardy, Durham, Dudgeon and Ross do not exactly correspond, possibly because different animals have been employed. But, generally speaking, small mononuclears, coarsely granular eosinophiles, and large hyaline mononuclear cells, probably endothelial in origin, are described.

As the result of experimental bacterial infection, Durham found a leukopenic stage lasting an hour, the normal hyaline and eosinophile cells disappearing from the peritoneal fluid, which becomes increased in amount, while the lymphocytes remain. The disappearance of the cells is due to their being collected into balls on the surface of the peritoneum, especially on the omentum, and not, as Metchnikoff believed, to their destruction. In very virulent infections the leukopenic stage may persist until death. According to Dudgeon and Ross² experiments the first cells found in the exudation are either small lymphocytes or coarsely granular eosinophiles; subsequently there are polymorphonuclear leukocytes (Metchnikoff's "macrophages," microxycytes) which are derived from the bloodvessels; these cells have been found by Beattie³ to diminish in number in non-fatal cases in from forty-eight to sixty hours after the onset, but to persist until death in fatal cases. They are active phagocytes, as are also the mononuclear cells (macrophages) derived from the endothelial cells of the peritoneum, which, however, according to Buxton,⁴ are unable to digest bacteria effectively in the absence of an early and marked polymorphonuclear reaction. Dudgeon and Ross' experiments on animals show that the coarsely granular

¹ *Transactions of the Association of American Physicians*, 1904, xix, 136.

² *British Medical Journal*, 1905, ii, 1043.

³ *Journal of Pathology and Bacteriology*, Edinburgh and London, 1903, viii, 129.

⁴ *British Medical Journal*, 1907, ii, 1422.

eosinophile leukocytes (megakaryocytes), which are usually regarded as non-phagocytic, are, in the early stages of peritoneal infection, among the most important if not the most important phagocytes.

The peritoneal exudation is not infrequently free from microorganisms and from phagocytes containing bacteria, while the surface of the intestine and the omentum shows their presence. This observation is important in the consideration of the question of chemical peritonitis, for it is clear that the peritoneal exudate may be sterile, and so suggest a non-bacterial peritonitis, because all the organisms have disappeared. In addition to the peritoneal and subperitoneal changes described above, the wall of the intestine shows small-celled infiltration around the vessels and fibrinous and serous exudation; the nerve cells of Meissner's and Auerbach's plexuses show degeneration. The solar plexus has also been found to show parenchymatous changes (Laiguel-Lavastine¹).

After-results.—An attack of acute peritonitis leaves behind it some slight thickening and opacity of the peritoneum; in cases in which blood has been extravasated pigmentation may be due to altered blood pigment. As the result of organization of fibrin, adhesions are commonly seen, but it is remarkable how completely adhesions may disappear in course of time.

Symptoms.—The signs and symptoms of diffuse peritonitis vary very widely in different cases; this depends on the cause of the peritonitis and on the virulence of the infecting organisms; in fact, different forms of peritonitis show somewhat analogous variations to those of pleurisy. It will, therefore, be best to give a very brief description of a common form of peritonitis, then to detail the manifestations that may occur in the various forms of peritonitis, and, lastly to refer to special forms, pneumococcal, gonococcal, and puerperal.

The course of diffuse acute peritonitis can be most satisfactorily sketched by a short account of an ordinary case of perforative peritonitis. There is a sudden onset with agonizing burning pain and a feeling that something has given away; this is rapidly followed by collapse. These symptoms are due to the perforation. The abdominal wall is rigidly contracted, and extremely tender. It is very rare to get tympanitic distention from escape of large quantities of gas into the peritoneal cavity at this stage. There is widespread and very severe pain which is exaggerated by any movement so that respiration is costal and shallow, and there is frequent vomiting. The collapse passes off in the course of a few hours, and for a time the patient appears fallaciously better; this period of latency or stage "of repose" (Symonds²) may be prolonged by the administration of opium or morphine. But if the disease be allowed to run its natural course, symptoms reappear and those of peritoneal inflammation become unmistakable; the abdomen gradually becomes distended, the legs being drawn up to relax the abdominal parietes, the arms usually lying above the head. There is extremely severe and constant pain, which becomes exaggerated in paroxysms and is made worse by the slightest movement, such as micturition; vomiting is frequent, the tongue becomes dry, the mouth parched, there is constant thirst, the temperature is somewhat raised, the pulse of high tension, wiry and rapid, the face pinched, anxious, and generally pale,

¹ *Arch. de méd. expér. et d'anal. path.*, Paris, 1905, xvii, 54.

² *British Medical Journal*, 1899, i, 520.

and its aspect that of grave toxæmia and of progressive exhaustion (*facies hippocratica*). As time goes on the abdominal distention increases, the pulse becomes thready, more rapid, and weaker, the skin is cold and clammy, and the extremities icy and blue, although the internal temperature may be considerably raised. Restlessness and anxiety persist, there is little or no sleep, the mind is usually clear to the end, but occasionally there is wandering or delirium. Pain, tenderness, and vomiting occasionally pass off shortly before death, which may be sudden from cardiac failure, but sometimes there is profuse vomiting just before death, and aspiration of the foul fluid into the lungs may be the last straw.

The Clinical Picture.—The underlying factors which are responsible for the clinical manifestations are:

1. Acute inflammation involving an extensive area.
2. Paralysis of the muscular walls of the intestines.
3. Septicæmia and toxæmia, due to the absorption of microörganisms and their toxins from the peritoneal surfaces and of toxins from the mucous membrane of the paralyzed intestines.
4. Abstraction of water from the tissues.

To go more into detail in the various clinical manifestations: The *onset* varies with the cause; when there is perforation of a hollow viscus, as in gastric or duodenal ulcer, the moment of the accident can usually be absolutely fixed. In cases of typhoid fever, however, perforation of an ulcer may not be marked by sudden and acute pain, and from dulling of the patient's powers of perception the existence of acute peritonitis may be an anxious problem. In patients in the late stages of renal disease, intra-abdominal new-growths, cirrhosis, chronic peritonitis, pulmonary tuberculosis, etc., the onset and even the existence of peritonitis may be latent and not suspected during life. In some cases, especially after abdominal operations and in intestinal obstruction, the onset of peritonitis is so gradual that it cannot be definitely fixed. In puerperal peritonitis and occasionally in slowly progressive forms of peritonitis, such as may occur in connection with some cases of appendicitis, the development is gradual and insidious.

With an acute onset there is more or less shock due to the damage done to the peritoneum by the sudden entry of the contents of an abscess or of a hollow viscus into the abdominal cavity. This shock may prove fatal before peritonitis has had time to develop.

The *facial aspect* of advanced peritonitis is characteristic, and has been described as "a composite photograph of hemorrhage, pain, and infection" (Crile). The face is pinched, the skin being tightly drawn over the underlying bones, the eyes sunken, and surrounded by dark rings, the skin gray or somewhat livid from failure of the circulation, and the expression anxious. This is the abdominal or Hippocratic face. In early stages, however, there may be nothing characteristic in the face, and the cheeks may be flushed.

The tongue, at first furred and moist, subsequently, from the loss of fluid, and absence of saliva, becomes shrivelled, dry, and stained by the vomit. Unless the mouth is carefully and constantly cleaned, sordes collect on the teeth. There is complete loss of appetite, and extreme thirst.

Position.—The patient lies on his back, the shoulders raised, and the thighs flexed so as to relax the abdominal walls as far as possible. The arms and head are tossed about and manifest the mental restlessness, but the rest of the body is kept as still as possible.

Pain is practically a constant symptom, and is therefore most important; it is only in cases in which a terminal peritonitis supervenes in the course of a chronic exhausting disease, such as carcinoma of the stomach or chronic renal disease, or of an acute disease, such as typhoid fever, in which the mental faculties are greatly dulled, that pain is absent.

The pain of peritonitis may be continuous with that of the causal perforation; in other forms of peritonitis it comes on more gradually, and steadily increases in severity until it becomes intolerable. It is constant, but becomes worse from time to time, the exacerbations probably depending on movements of the intestines; vomiting, cough, or any kind of movement makes the pain worse, and accordingly the patient keeps the abdomen and trunk absolutely still, and holds the arms above the head, while the movements of the diaphragm are greatly restricted.

The pain is general all over the abdomen, but may be more marked in some one part, such as the umbilicus or the right iliac fossa, and sometimes this may correspond with the starting point of the disease. But this may be fallacious; for example, the pain may be most marked in the appendix region in perforated duodenal ulcer, and in pelvic appendicitis the pain is not uncommonly referred to the umbilicus. Toward the end the pain may disappear; this may be in part the result of the effusion and of intestinal paralysis, which prevents the intestines from rubbing against the parietal peritoneum, in part of the continued effect of toxæmia on the exhausted sensorium. The diminution or disappearance of pain, therefore, does not mean that there is any improvement.

Tenderness.—There is no true cutaneous tenderness or pain in acute peritonitis (Head¹); if present it is due to some definite visceral lesion; thus, although the zone of cutaneous hyperæsthesia due to appendicitis may disappear when perforation into the general peritoneal cavity occurs (Sherren²), it may persist when peritonitis has supervened; out of 8 cases of peritonitis due to appendicitis, especially investigated with regard to this point by Dr. H. Robinson, at St. George's Hospital, three showed an area of cutaneous hyperæsthesia. In order to test accurately for the presence of cutaneous hyperæsthesia, the skin, without any of the underlying tissues, should be carefully picked up between the finger and thumb. In acute peritonitis the abdominal reflexes are not exaggerated, as they would be if the tenderness were cutaneous (Head). Tenderness of the deeper tissues is due to peritonitis and implication of the parietal peritoneum and its somatic nerve supply in the inflammatory process. The tenderness gets progressively worse, and may be so extreme that the patient cannot bear the weight of the bed-clothes; it is universal and corresponds with the extent of the peritoneum, being elicited by rectal and vaginal examination. On rectal examination, ballooning of the rectum may be found.

Rigidity of the Abdominal Walls.—In the early stages the muscles are firmly contracted from spasm, and there may even be retraction; this is most marked in cases of perforative peritonitis. In strong and muscular men the rigidity is prominent and lasts for a considerable time; in fact, in very acute cases it may persist until the end. In women with very flaccid abdominal parietes there may be no appreciable rigidity. Although this muscular defence is one of the most common events in the course of acute peritonitis,

¹ *Brain*, 1893, xvi, 94.

² *Lancet*, 1903, ii, 816.

it may be absent throughout; this is seen in gonococcal peritonitis, and even in cases in which there is no special atrophy of the abdominal muscles. This absence of rigidity is more likely to occur in non-perforative cases and in the somewhat slowly progressive fibrinopurulent peritonitis described by Mikulicz. As a result of this muscular spasm, the abdomen becomes more or less immovable; this is a valuable sign of peritonitis, but movement of the abdomen does not prove that peritonitis is absent. Muscular rigidity passes off when the patient is anesthetized.

Tympanites.—Except in the fulminating and rapidly fatal cases, the early rigidity gradually passes off, and in about forty-eight hours after the onset in acute cases is succeeded by abdominal distention; in less acute cases this may be delayed for a day or two longer. The abdominal distention may come on rapidly after rupture of the stomach and escape of gas into the abdominal cavity.

The tympanites or meteorism of peritonitis is due to distention of the paralyzed intestines with flatus; to a slight degree accumulation of liquid feces in the dilated intestines and inflammatory peritoneal exudate, which is seldom large in amount, contribute to the distention. Its onset is naturally favored by a flaccid condition of the abdominal parietes, such as may result from parturition, abdominal tumors, or ascites. The abdomen becomes prominent, hyperresonant, and the skin tight, stretched, and shiny. Abdominal movement is abolished.

The distention pushes up the liver in such a manner that the normal relation of that organ to the parietes is altered, and very little of it remains in contact with the body wall; in extreme instances the thin edge of the liver looks upward and backward instead of downward and forward. As a natural consequence, the hepatic dullness is diminished or obliterated, and this without any free gas in the abdominal cavity. Absence of liver dullness must not be taken as evidence of perforative peritonitis. The diaphragm is pressed up, so that the lower lobes of the lungs become cedematous and partially collapsed, and the breathing thus further embarrassed. The apex of the heart may be somewhat displaced upward, into the fourth intercostal space.

The intestinal paralysis, which is one of the most important factors in acute peritonitis, is probably nervous and due to reflex shock; it may be increased by cedema and inflammation of the muscular walls and the local action of toxins on the neuromuscular apparatus of the intestinal wall. But from the rapid onset of distention in some cases of peritonitis, before inflammation can have spread sufficiently into the intestinal wall to produce muscular paralysis, this is certainly not the only factor concerned.

Distention is lessened by copious vomiting. The distention may force a coil of intestine into an old hernial sac, and so suggest that the symptoms are due to strangulation of the bowel, but examination will show that the hernia is reducible.

Percussion is painful, and therefore should be light; when there is distention the note is usually tympanitic, but in certain areas, especially the flanks, there may be dullness due to collections of peritoneal exudate or to coils of intestine containing fluid feces. In rare instances a friction rub can be heard. On the other hand, a valuable sign of intestinal paralysis is the entire absence of any gurgling sound when the abdomen is auscultated for five minutes at a time (Greig Smith).

Edema of the abdominal wall is extremely rare; when it does occur it is usually in connection with a localized collection of pus, for example, appendicular abscess. It is not a sign of diffuse peritonitis.

Vomiting is one of the most characteristic and, next to pain, the most constant manifestation. It may be absent, however, especially in young children. In the early stages, of which it is often the first symptom, it is probably a reflex result of irritation of the peritoneum, although it has been regarded as due to the action of toxins on the medulla. In the later stages it is, like the vomiting of acute obstruction, due to a reflux flow of fluid into the stomach from the paralyzed intestines. That it depends on paralysis of the intestine, and overflow into the stomach of their stagnating contents is supported by the rule that it is less prominent or absent in cases in which the bowels act or in which there is diarrhoea, as in puerperal peritonitis; further, washing out the stomach prevents the vomiting. It is commonly excited by taking fluid to relieve the urgent thirst.

The vomited materials are first those of the stomach, then those of the upper part of the small intestine containing easily recognizable bile, and lastly feculent or stercoraceous matter of an extremely offensive character. In severe cases the vomit is often brown or dark in color from the presence of blood. The fluid is brought up with comparatively little effort, welling up into the mouth. The amount varies somewhat, but is much less than in mechanical obstruction. It is rather remarkable that vomiting occurs in cases of perforation of the stomach; it might have been surmised that continued vomiting would militate against a diagnosis of perforated gastric ulcer, but this is not so. Thus the Fenwicks found vomiting in 29 per cent. of perforated gastric ulcer, Finney in 40 per cent., and English in 74 per cent. The movement entailed in vomiting exaggerates the abdominal pain. Nausea is common, there is a bad taste in the mouth, and foul gas may be belched up. In the later stages retching may be substituted for vomiting. The loss of fluid entails continual thirst.

Hiccough, when it occurs, usually comes on in the late stages of peritonitis when the abdomen is considerably distended. It is a reflex phenomenon due to implication of the phrenic nerve.

Rigors are not common; they are seen in some cases of puerperal peritonitis, and in the prolonged form of progressive fibrinopurulent peritonitis.

Constipation is the rule, and, from intestinal paralysis, is often so marked, both for flatus as well as for faeces, as to give the case the aspect of acute mechanical obstruction. An enema will, however, usually bring away some fecal matter in acute peritonitis without any improvement in the patient's condition; this does not hold good for mechanical obstruction. So constant is constipation in acute peritonitis that cases in which it is absent or in which the bowels can be made to act may generally be regarded as hopeful. In puerperal peritonitis, however, diarrhoea, due presumably to some peculiar form of toxin, is the rule. In other forms, except in that due to pneumococcal infection, it is exceptional.

Respiration.—As diaphragmatic and abdominal movements are greatly reduced or abolished, the respiratory excursions mainly depend on the intercostal muscles. The upper parts of the lungs are therefore employed to a larger extent as a means of compensation for the impaired movements of the lower lobes, and some of the extraordinary muscles of inspiration come into action. The respirations are painful, shallow, and considerably

increased in rate (40 to 50 per minute); coughing and deep breaths are restrained.

The pulse varies in character at different stages of the disease. It is nearly always regular, quickened, and becomes progressively more rapid, being 100 to 120 in the early stages, and becoming running, 170, or uncountable, toward the end. In some instances the rate of the pulse may be little if at all increased, although there is extensive purulent peritonitis; this has been ascribed to interference with toxic absorption from blocking of the stomata and lymphatics, but is not necessarily a sign that the patient is improving. There is no correspondence between the rate of the pulse and the temperature; the temperature may be low and the pulse extremely rapid. The pulse is small and hard in the early stages, and is spoken of as wiry; Crile,¹ who found the average blood pressure in 20 cases to be 166 mm. of mercury, ascribes the small volume to the accumulation of blood in the splanchnic area and the hardness to increased blood pressure induced by reflex stimulation of the vasomotor centres from the peritoneum. In collapse and in the later stages the blood pressure falls and the pulse becomes thready and eventually perhaps imperceptible.

The temperature in peritonitis is so extremely variable as to be of little or no value in diagnosis or as to the degree and gravity of the peritonitis. In peritonitis there is usually a more considerable difference between the rectal and the surface temperatures, such as the axillary, than in other diseases; this is connected with the stagnation of blood in the splanchnic area and the correspondingly small amount of blood in the cutaneous vessels.

In cases of perforative peritonitis the initial shock nearly always depresses the temperature; this is most noticeable in typhoid fever, in which the writer has seen a fall of eight degrees. But this is not an absolute rule, the variation probably depends on how much of the peritoneum is inundated by the contents of the bowel. It is possible that the form of infection may have something to do with the temperature, and that a particularly virulent microbic invasion may so paralyze the power of resistance that there is less febrile reaction than in a less severe form of infection. In the vast majority of the cases of diffuse peritonitis the temperature is raised in some period of its course; it may be raised continuously, usually not to any great extent, and very seldom above 104°. A relatively high temperature, especially in the early stages, is more likely to occur in healthy and vigorous individuals. A persistently low temperature may depend on the severity of the infection and on failure of the organism to react, and is therefore a grave sign.

Blood.—There is anæmia, but the erythrocyte count is not very greatly diminished; probably the concentration of the blood accounts for this. In 16 cases examined by Da Costa, the average was 3,970,000 reds, and the color-index 0.78. In fulminating cases in which the resisting powers of the body are paralyzed by the intensity of the toxæmia, leukocytosis is absent and there may be leukopenia. In less severe peritonitis there is a leukocytosis which in some instances may be extremely marked. It is of the ordinary polymorphonuclear neutrophile form. According to Locke² and Gulland³ the glyco-genic reaction seen in the polymorphonuclear neu-

¹ *Blood Pressure in Surgery*, Philadelphia, 1903.

² *Boston Medical and Surgical Journal*, 1902, cxlvii, 287.

³ *British Medical Journal*, 1904, i, 880.

trophiles is much more trustworthy, for it is present in cases of peritonitis with such a "brutal" infection that leukocytosis does not appear. Cabot¹ lays stress on the importance of the increase of the fibrin network (hyperinosis) in the diagnosis from mechanical obstruction, for this increase does not occur in obstruction, while leukocytosis does.

Blood Cultures.—Although the fatal results of peritonitis are usually considered to depend upon septicæmia as well as on toxæmia, and although organisms can often be found after death in the heart's blood, it does not appear that the presence of organisms in the blood during life has been proven. In this connection it may be pointed out that in human septicæmia pyrogenetic cocci are demonstrated with difficulty in the blood, thus contrasting with experimental peritoneal infection and septicæmia in animals. Libman² found that in 25 cases of peritonitis, mainly of intestinal origin, the blood was sterile, although in some instances the blood cultures were made shortly before death.

The urine is scanty, high colored, and may contain a small quantity of albumin, diacetic acid, and acetone. The most important point, however, is the large quantity of indican, which Nothnagel regards as practically constant and as present in larger amounts than in any other disease except intestinal obstruction. The normal amount in adults is from 5 to 20 mg. in the twenty-four hours, but in pathological conditions it may be increased up to 150 mg. Indicanuria takes some twelve to twenty-four hours to appear after the onset of perforative peritonitis.

According to Lennander, the urine contains the organisms responsible for the peritonitis. Their presence, especially as he states they may appear within twenty-four hours of the onset of the disease, may possibly in the future be of some value in directing the treatment. Unfortunately, as regards gonococcal peritonitis, the presence of gonococci in the urine could hardly be regarded as an infallible guide to the nature of the peritoneal infection, as there might be a mixed infection.

From the presence of peritonitis over the bladder micturition is painful, and usually urine is retained, as if from paralysis of that viscus resembling that of the intestines. Occasionally, on the other hand, micturition is more frequent than normal.

The mental faculties are often clear to the end, but sometimes they are somewhat obscured by the progressive toxæmia. In cases of peritonitis supervening in the course of typhoid fever the mental obscuration due to the primary disease may be expected. Sleeplessness is necessarily the rule, headache is common, and great anxiety and a painful form of restlessness are often present.

Death may come quite suddenly from cardiac failure brought about by the action of toxins on the heart muscle, and may follow almost immediately after copious vomiting. Death sometimes comes gradually from progressive asthenia.

The duration is very variable and depends on the cause and on the nature of the infection, the previous state of the patient, young and vigorous, or old and the subject of some exhausting disease, and on the treatment. Thus, in the most virulent and fulminating forms, as in some cases of

¹ *Examination of the Blood*, 1904, p. 282.

² *Johns Hopkins Hospital Bulletin*, 1906, xvii, 221.

appendicular and puerperal infection, death may occur in thirty-six to forty-eight hours from the onset, whereas the more usual and less severe forms generally last four, five, or even more days. A previously healthy boy may live through several days with severe peritonitis, while a patient with carcinoma of the stomach may only survive the onset of perforative peritonitis for eighteen to twenty-four hours. In pneumococcal peritonitis the duration is very considerably prolonged, and, as pointed out elsewhere, the peritonitis may become localized into residual abscesses, so that the symptoms abate without disappearing. In the progressive fibrinopurulent peritonitis, described by Mikulicz, which constitutes a transition from circumscribed to diffuse peritonitis, the disease may go on for weeks.

Pneumococcal Peritonitis.—This is a rare form, but presents some rather special features which necessitate a separate description. In 140 cases of peritonitis examined bacteriologically, Netter found this form only twice. Jensen¹ has collected 106 cases. The disease is much commoner in children than in adults. It may occur very early in life; Gossage² reported 4 fatal cases of primary pneumococcal peritonitis in infants under three months, and Dudgeon and Sargent report its occurrence in a male infant aged seven weeks. Under the age of fifteen years it occurs more often in girls than in boys, according to Annand and Bowen,³ who have collected 91 cases, in the proportion of 3 to 1, but after that age its incidence is equal in the two sexes.

Pneumococcal peritonitis may be primary, secondary, or, as Bowen and Annand point out, it may be difficult from the rapid spread of the infection to decide whether it is primary or secondary. The following channels have been regarded by various authors as enabling the pneumococci to reach the peritoneum: (1) Through the blood stream, the organisms being absorbed from foci of pneumococcal infection in the thorax or possibly from the throat or ear; (2) from the stomach (pneumococcal gastritis), intestines in inflammatory conditions, appendicitis, or foci in the abdominal viscera; (3) from the pleuræ through the diaphragm; and (4) through the Fallopian tubes. In favor of the last path, the greater frequency of the process in the lower half of the abdomen and its preference for young girls were at one time urged. Against this hypothesis it has been argued that there is generally no evidence of vulvovaginitis clinically or at autopsy. Bond's⁴ observations on ascending mucous currents in the uterus and Fallopian tubes, however, make it probable that the pneumococcus may be rapidly carried up the genital passages in a few hours, so that there is not sufficient time for local inflammation to become evident. Probably infection of the peritoneum occurs by different channels in different cases. There may be associated pneumococcal lesions elsewhere, such as pneumonia, empyema, broncho-pneumonia, pericarditis, otitis, but in the majority of instances the peritoneum is the first part attacked. Diffuse peritonitis is rare in pneumonia; in 4454 cases⁵ of lobar pneumonia in patients over ten years of age in the hospitals of London there were 11 fatal cases, or 0.25 per cent. Pneumococcal infection of several serous membranes, the pleura, pericardium, and peritoneum, pneumococcal polyorrhymenitis, may occur, especially in young

¹ *Arch. f. klin. Chir.*, 1903, xx, 91.

² *Proceedings of the Royal Society of Medicine, Medical Section*, London, 1908, i, 64.

³ *Lancet*, 1906, i, 1591.

⁴ *British Medical Journal*, 1906, ii, 1689.

⁵ Statistics of Hadley, Pasteur, Fawcett, Owen, Gossage, *Proceedings of the Royal Society of Medicine, Medical Section*, London, 1908, i, 61 et seq.

children. In most cases the peritoneum has been previously healthy, and there is no obvious reason why it has been picked out for infection; in a few instances there has been ascites due to cirrhosis or to chronic nephritis (Sevestre and Aubertin¹). Pneumococcic peritonitis was primary, that is, the chief or only focus of pneumococcal infection in 47 out of 74 cases, and in 34 of these 47 cases the peritonitis was encysted (Lenormant and Lecène²).

In about half the cases the peritonitis is encysted or localized; in the other half it is diffuse or widespread. A localized abscess may either be primary or a result of diffuse pneumococcal peritonitis. The exudate is like that of a pneumococcal empyema; it is highly fibrinous and prone to coagulate, yellowish green in color, and odorless. The character of the exudation renders thorough drainage of the peritoneum difficult. In some instances, however, a much more fluid exudate has been found. When the peritonitis is localized—the abdominal empyema of Lennander—an abscess results which is usually below the umbilicus, and is particularly prone to point at the umbilicus.

Clinically the disease has three stages: (1) Without any prodromal symptoms there is an acute onset with vomiting, followed by signs of peritonitis. As the inflammation is often mainly in or near the pelvis, appendicitis is not uncommonly diagnosed. (2) After a few days the symptoms subside, the temperature falls, but diarrhoea is usually present, and for this reason typhoid fever may be suspected. (3) A prolonged stage in which residual abscesses may form, usually in the upper part of the abdomen, giving rise to continued fever and imitating tuberculous peritonitis. As bearing on the resemblance of the manifestations to those of pneumonia, it may be mentioned that herpes labialis has been noted in some cases and that the temperature occasionally falls by crisis.

It is one of the less severe forms of peritonitis; the *prognosis* in primary encysted pneumococcal peritonitis is fairly good, but it is less favorable in the diffuse form; thus, Bowen and Annand found that recovery occurred in 86 per cent. of the encysted and in only 14 per cent. of the diffuse form.

Gonococcal Peritonitis.—As the result of comparatively recent work, especially that of Cushing³ and Hunner,⁴ it is clear that the gonococcus alone, *i. e.*, in pure culture, is capable of setting up diffuse peritonitis. The peritonitis is not, however, of a severe character, and it has been suggested that this is because the gonococcus flourishes best at from 91° to 98° F., or about the normal temperature of the body, and that a higher temperature, such as would be met with in the peritoneal cavity, inhibits its activity (Hale White⁵). When the normal resistance of the peritoneum is diminished by menstruation or by cold, gonococci in the Fallopian tubes have a better chance of infecting the peritoneum. Gonococci enter the peritoneum from the ostia of the Fallopian tubes, but gonococcal peritonitis has followed leakage from a pyosalpinx during removal by operation. In males infection is said to travel up the lymphatics of the spermatic cord.

The disease is very rare in men and its frequency in women has not yet

¹ *Bull. et mém. Soc. méd. d. hôp.*, Paris, 1906, xxiii, 215.

² *Rev. de gyn. et de chir. abdom.*, Paris, 1905, ix, 225.

³ *Johns Hopkins Hospital Bulletin*, 1899, x, 75. (This article contains the detailed history of gonococcal peritonitis.)

⁴ *Ibid.*, 1902, xiii, 247.

⁵ *System of Medicine* (Allbutt and Rolleston), 1905, i, 855.

been established by extensive statistics; but diffuse gonococcal peritonitis is not common. In 1907 Goodman¹ collected 75 cases, but only 30 of these had been established by bacteriological examination at autopsy or operation. It may attack girls under puberty (Northrup,² Comby³), and it is highly probable that it is often overlooked or not recognized. It usually follows or precedes menstruation, and may appear shortly after delivery. The anatomical lesions, as pointed out by Cushing, are comparatively slight; there is a dry, fibrinous exudate without pus or serum, and the peritoneum is uniformly injected in the diffuse cases.

Clinically, gonococcal peritonitis comes on suddenly with great severity, which usually becomes greatly mitigated in twenty-four or forty-eight hours. Comby speaks of the condition as one of peritonism rather than of peritonitis. It appears to be more fatal in children than in adults; the mortality in children has been estimated at 20 per cent. It may imitate appendicitis, as it mainly affects the lower part of the abdomen. Abdominal distention is usually not marked, and muscular rigidity is absent. The existence of gonococcal peritonitis should be suspected in the presence of gonorrhœa when the peritonitis is mainly in the lower part of the abdomen. At the onset of the disease the gonorrhœal discharge may be slight, and as its existence is often firmly denied careful examination is necessary to avoid overlooking it. It is usually agreed that the patients should not be operated upon, but in 20 cases in which laparotomy was performed there were four deaths only (Goodman). The difficulty is to decide in a given case of a woman with a vaginal discharge containing gonococci whether the peritonitis is due to a pure infection or to a mixed infection, such as may result from rupture of a pyosalpinx.

Puerperal Peritonitis.—The infection gains entry through tears or wounds of the genital passages, and is much commoner in primiparæ than in multiparæ. It is not uncommon in criminal abortion. The infection spreads from the genital organs by the lymphatics, and may be localized or widespread in the peritoneal sac. The organism is most commonly *Streptococcus pyogenes*, but staphylococci or the gonococcus may be the causal agent. The exudation may be purulent, sanious, or putrid.

Clinically, puerperal peritonitis is characterized by the following features: there is grave intoxication, usually diarrhœa; vomiting is less prominent than in other forms; meteorism is marked, partly no doubt from the weakened and relaxed condition of the abdominal walls; and a high mortality, which may be correlated with the presence of *Streptococcus pyogenes*. At its onset the lochia may be noticed to be offensive and the mammary secretions to be suppressed. Complications are prone to occur, and include septicæmia, pyæmia, empyema, pericarditis, arthritis, and phlebothrombosis.

Diagnosis.—In characteristic cases the association of the prominent symptoms of acute pain, tenderness, distention, and immobility of the abdomen, vomiting, constipation, prostration and collapse, together with the history, make the existence of acute peritonitis clear. But in many cases the

¹ *Annals of Surgery*, 1907, xlv, 111.

² *Archives Pediatrics*, New York, 1903, 910, and *Transactions of Association of American Physicians*, 1903, xviii, 203.

³ *Arch. mal. d. enfants*, 1901, iv, 513.

decision is far from easy; when peritonitis supervenes late in the course of chronic nephritis it may be almost latent, when it occurs in typhoid fever it may be difficult to distinguish from tympanites, and in acute intestinal obstruction its manifestations are combined with and obscured by those of the primary disease. Further, the symptoms may be almost entirely masked by opium or morphine. In some cases the majority of the signs and symptoms may be absent, although there is advanced peritonitis. This masked or latent form of peritonitis may occur in patients in the last stages of exhausting diseases, such as cancer. After operations on the abdomen the only evidence of peritonitis may be vomiting and a rapid pulse.

Diagnosis of Acute Peritonitis from Conditions Simulating It.—In hysterical subjects a condition of peritonism may occur and give rise to considerable anxiety, as the diagnosis between it and genuine peritonitis occurring in an hysterical patient may be difficult. The history of the patient and any concomitant nervous symptoms must be taken into account. As an important point in the history, the occurrence of exactly similar attacks in the past should be specially noted. In such cases there is cutaneous hyperæsthesia, deep tenderness is not more marked than the superficial hyperæsthesia, and may disappear if the patient's attention be distracted.

Severe colic, whether due to lead, constipation, or the passage of a gall-stone or renal calculus, may to some extent imitate acute peritonitis, since the exacerbations of pain in peritonitis are probably of the same nature. In colic there is, as a rule, no real tenderness, and the pain may be relieved by pressure. In severe lead colic there may, however, be tenderness, but, as T. Oliver¹ has pointed out, the pupils are often unequal and there may be tenderness over the vagus in the neck. In a doubtful case with abdominal tenderness the possibility of lead poisoning should always be considered. In colic due to the passage of a calculus vomiting occurs, but it is rare in the other forms. The history of the case and examination for evidence of lead poisoning, renal, or hepatic disease should be carefully considered. A raised temperature at the onset is strongly in favor of peritonitis and against ordinary colic. Colic, however, is more likely to be confused with the causes of peritonitis—such as appendicitis—than with the disease itself. The rare and puzzling condition of enteralgia may also imitate peritonitis; here again the history of repeated similar attacks is of importance. Extremely acute enterocolitis is accompanied by the same collapse as in peritonitis, but there is marked diarrhœa and the pain is of a colicky character.

Tympanites should be readily distinguished from acute peritonitis by the absence of tenderness and vomiting.

In acute mechanical intestinal obstruction acute peritonitis supervenes after some three or four days, and, conversely, acute peritonitis produces intestinal paralysis and the symptoms of obstruction, so that the diagnosis as to the primary lesion may be difficult. In acute obstruction the temperature is not raised, and the abdomen is not tender or rigid, before the onset of secondary peritonitis. In mechanical obstruction fæces and wind are not passed, while in peritonitis an enema will often bring some away. In mechanical obstruction vomiting is more profuse, the pain is colicky and not continuous, as in peritonitis.

¹ *System of Medicine* (Allbutt and Rolleston), 1906, ii, part i, 1045.

Extensive hemorrhage into the peritoneum, usually due to rupture of an ectopic gestation, may imitate perforative peritonitis very closely; in fact, the manifestations in both instances are those of shock. A similar clinical condition of peritonism may follow hemorrhage into the mesentery, torsion of an undescended testis, of an ovarian cyst, of a floating kidney, or of a wandering spleen. In a woman likely to be pregnant this train of symptoms, especially the anæmia progressing until it becomes extreme, should suggest the possibility of rupture of an ectopic gestation, while the presence of an ovarian tumor should arouse a suspicion of torsion. The distinction, however, is hardly essential, for laparotomy is the proper treatment in either case. Rupture of an abdominal aneurism and embolism of the superior mesenteric artery have been known to imitate acute peritonitis (Osler).

Acute hemorrhagic pancreatitis may imitate acute perforative peritonitis; great collapse, abdominal distention, complete constipation, and vomiting follow the sudden onset of pain in the epigastric region. If the abdomen be opened, turbid fluid and fat necrosis may be found. It may be noted, however, that fat necrosis may be due to the escape of pancreatic fluid through the perforation of a duodenal ulcer,¹ so that its discovery, although almost pathognomonic of pancreatitis, does not absolutely exclude perforation. In pancreatitis there is extreme feebleness of the pulse, the temperature is often not raised, and the symptoms are more suggestive of mechanical obstruction than of peritonitis.

Acute inflammatory conditions in the thorax, such as lobar pneumonia or pleurisy, may, especially at the onset before physical signs have appeared, imitate peritonitis. The sharp pain of pleurisy, the onset of which may wake a patient up in the night, has been mistaken for perforation of a gastric ulcer or of a gangrenous appendix. Difficulty is especially likely to arise in children in whom the appearance of the physical signs of pneumonia, particularly when apical, is often much delayed. Thus pain referred to the right iliac fossa may imitate acute appendicitis, and the fever, constipation, and abdominal distention suggest peritonitis. It is, therefore, most important to examine the chest, to note the respiration rate and temperature, which are higher and more persistent in pneumonia than in peritonitis, and to inquire for cough. In pneumonia the respirations may be jerky and grunting. The rigidity of the abdominal muscles sometimes seen in pneumonia, especially in children, will be found to relax between the respirations, and may even disappear on prolonged and deep pressure (Clogg²), while the referred pain of thoracic disease is mainly superficial and not increased on deep pressure, as in peritonitis. The temperature may be of some assistance; low at the onset of perforative peritonitis, it then rises, but may fall after some hours, although the patient's condition is not improved, it thus differs from the continuously raised temperature of pneumonia. Indican in the urine is in favor of peritonitis. Tuberculous peritonitis in rare cases comes on with such acute symptoms in children as to imitate very closely peritonitis due to appendicitis; this has been thought to be due to thrombosis of the mesenteric vessels (R. Johnson³).

In women about the menstrual period sudden acute pain may imitate perforation of a hollow viscus or rupture of an ectopic gestation. The symp-

¹ J. D. C. White, *Archives of the Pathological Institute of London Hospital*, 1906, i, 48.

² *Clinical Journal*, London, 1906, xxix, 93.

³ *Practitioner*, London, 1906, lxxvi, 332.

toms pass off, as a rule, but are alarming enough at the time. The underlying cause has been thought to be hemorrhage into the ovary. In one case attended by great temporary collapse the real cause appeared to be synthetic drugs taken to relieve the pain on the first day of the period.

Uræmia may imitate diffuse peritonitis, and it must be remembered that uræmic ulceration of the intestine may lead to perforative peritonitis, and that hæmatogenous peritonitis may supervene in chronic renal disease. The character of the urine, the pulse, the presence of œdema, and the history should lead to the recognition of renal disease.

In very rare instances acute abdominal symptoms imitating appendicitis or even peritonitis supervene in the course of Addison's disease (Nattan-Larrier¹) and appear to be allied to the vomiting commonly seen in that disease. It is important to remember this rare manifestation of suprarenal inadequacy, as a fruitless laparotomy would be very likely to prove rapidly fatal.

In severe pernicious malaria, due to æstivo-autumnal parasites, symptoms suggesting peritonitis may occur; this, of course, only occurs in malarial districts. The diagnosis depends on the detection of the parasite in the blood.

When the presence of peritonitis has been determined it is most important to diagnose the cause and to decide whether it is due to perforation of a gastric, duodenal, or an intestinal ulcer, to appendicitis, to rupture of an abscess, and, if so, where, to gonococcal or pneumococcal infection, and so on; for on the correct diagnosis of the cause depends the site of the abdominal incision in the first instance. As bearing on the cause of peritonitis in any given cause the following points may be taken into consideration:

Age.—In children peritonitis of sudden onset is most commonly due to fulminating appendicitis; but although far from common, pneumococcal and gonococcal peritonitis must be considered.

Sex.—In young women perforated gastric ulcer and pelvic infection are the most common causes.

History.—Symptoms of dyspepsia in young women at once suggest a perforated gastric ulcer, while in patients at or about fifty years of age carcinoma of the stomach with perforation should not be forgotten. When peritonitis comes on suddenly in a patient who has had gastro-enterostomy done for gastric ulcer, the possibility of perforation of a peptic jejunal ulcer should be thought of. The existence of typhoid fever, advanced kidney disease, or of a pelvic abscess will indicate the probable starting point of the peritoneal infection.

The position in which the pain is first felt should be considered, but it is by no means a certain guide, for, although pain starting in the right iliac fossa naturally suggests appendicitis, pain in this situation may be due to perforation of a gastric or duodenal ulcer.

The sudden onset of peritonitis in a person in good health up to that time may be due, especially in children, to fulminating appendicitis, in young women to perforation of a latent gastric ulcer, and in men to perforation of a duodenal ulcer which is relatively more often latent than a gastric ulcer is in women.

¹ "Le syndrome pseudo-appendiculaire et les accidents simulant la péritonite dans la maladie d'Addison," *Clinique médicale de l'Hôtel-Dieu*, Paris, 1906, v, 250.

Prognosis.—The prognosis of acute diffuse peritonitis is undoubtedly very bad. Statistics differ very much in the percentages of mortality, because, as has been pointed out, a case which at operation one surgeon may describe as diffuse another would regard as general or universal. In 100 cases at the London Hospital there were 70 deaths and 30 recoveries (Treves), but the different forms of peritonitis vary so greatly in their severity that no conclusion of any value can be obtained from statistics in which cases of the various kinds are indiscriminately included.

Other things being equal, peritonitis is more fatal at the two extremes of life. The previous health of the patient, the presence of disease elsewhere, cachexia, and alcoholism, of course, have a bearing on the prognosis.

As the fatality of peritonitis depends on toxæmia and septicæmia, the prognosis to some extent varies with the degree of the toxic symptoms; the cases of acute peritoneal sepsis in which there is very little reaction on the part of the peritoneum are almost always fatal. Bad as the prognosis is in purulent peritonitis, it occasionally happens that the inflammation becomes localized and an abscess results.

A very rapid pulse, a persistently low temperature, abdominal distention, and absence of leukocytosis are of bad prognosis.

From a bacteriological point of view peritonitis due to Dudgeon and Sargent's white staphylococcus is the most benign form, whilst that due to gonococci is comparatively mild. In pneumococcal peritonitis the prognosis is graver than in the forms just referred to, but is not so serious as in those now to be mentioned. The commonest cause of peritonitis is infection with virulent colon bacilli; if present alone in the peritoneal cavity at a distance from the infecting focus the outlook is bad; and if all the phagocytes are degenerated the prognosis is practically hopeless. If *Bacillus coli* and the white staphylococcus are associated, the outlook is not so bad, but is still very serious. The *Streptococcus pyogenes* gives rise to the most acute and fatal peritonitis, and it is doubtful whether any surgical procedure offers a prospect of success. Peritonitis due to the *Bacillus pyocyaneus* is hardly less fatal (Dudgeon and Sargent).

The prognosis is almost necessarily fatal in cases of perforative peritonitis unless operated upon; when operation has been performed the outlook is much better in cases of traumatic rupture or bullet wounds of the intestine than in perforation due to disease, such as typhoid fever or appendicitis, for in the latter conditions the virulence of *Bacillus coli* is much increased. Similarly the prognosis is better in cases of perforation of a gastric or duodenal ulcer than in perforation of an intestinal ulcer lower down, because, as shown by Cushing and Livingood,¹ the number of colon bacilli steadily diminishes from the ileocaecal valve to the stomach, and the duodenum is often sterile when empty. The interval between the onset of peritonitis and the time at which the operation is performed has an important bearing on the prognosis; operation within twelve hours of the onset should be successful, while an interval of twenty-four hours makes the outlook very grave.

Treatment.—Prophylaxis.—Under this heading the briefest reference only need be made to the desirability of preventing or curing the causes of general peritonitis, such as gastric or intestinal ulceration, appendicitis,

¹ *Contributions to the Science of Medicine by the pupils of W. H. Welch*, 1900.

pelvic inflammation, and intra-abdominal abscesses. Among these the importance of timely surgical interference in acute appendicitis cannot be exaggerated. It is almost unnecessary to insist that strict precautions should always be taken to avoid the introduction of organisms into the peritoneal cavity when the abdomen is tapped for the relief of ascites or when laparotomy is performed. The surgical technique has greatly improved in recent years and has exercised a beneficial effect in lowering the incidence of post-operative peritonitis.

Various prophylactic measures have been adopted in order to immunize the peritoneum against probable infection during the course of laparotomy. These measures are mainly based on Issaëff's¹ observation that injection of saline and other fluids into the peritoneal cavity produces a local leukocytosis, and so protects the animal against some forms of microbial infection; this "Issaëff's resistance period" lasts four or five days. As pointed out, Buxton and Tracy² consider the protection mainly mechanical and due to blocking of the lymphatics by the inflammatory products, by means of which bacteriemia is prevented. Schmidt,³ who injected sterilized horse-serum into the peritoneal cavity, through a trocar especially constructed by Petit, seventeen or nineteen hours before the operation, with the view of producing a local leukocytosis in the peritoneal sac and so providing for phagocytosis of any bacteria that might escape into it during the course of the operation, obtained encouraging results; this procedure might be used in cases of appendicitis, so as to prevent peritonitis. Mikulicz injected nuclein twelve hours before operation, to bring about the same result. On the ground of the terrible mortality in cases of operation for perforation in typhoid fever, it has been advocated that, instead of laparotomy, when symptoms suggesting perforation arise, a 1 per cent. solution of nucleinate of soda should be injected hypodermically in the flank or thigh in order to induce an antagonistic leukocytosis; about 40 cc. are given at each injection (Chantemesse and Kahn⁴). The danger of injecting fluids such as serum or physiological salt solution through the abdominal wall into the peritoneal cavity is that the viscera may be wounded and acute peritonitis started. The introduction into the abdominal cavity, at the end of operations on cases of peritonitis, of hot horse-serum or salt solution has been advocated in order to increase leukocytosis; about 30 cc. of sterilized horse-serum is poured through the drainage tube, and has given extremely good results (Petit⁵).

Exner⁶ has suggested that before operations on peritonitis adrenalin should be injected into the peritoneal cavity, in order to prevent absorption of toxins by the lymphatics.

Surgical Treatment.—The great majority of cases depend on infection from some local focus in the abdominal cavity, and are therefore rightly treated by removal of the cause. In the small minority of cases of general peritonitis in which the infection is hæmatogenous or brought by the blood-vessels, operative interference is not so necessary, but is required to let out a local abscess resulting from the peritoneal inflammation, as may be seen in pneumococcal peritonitis.

¹ *Ztschr. f. Hyg.*, Tübingen, 1894, xvi, 287.

² *Journal of Medical Research*, Boston, 1907, xvii, 1.

³ *Deutsche med. Woch.*, 1904, 1806.

⁴ *Bull. Acad. de méd.*, Paris, 1907, lvii, 736.

⁵ *New York Medical Record*, 1907, lxxi, 1017.

⁶ *Ztschr. f. Heilk.*, 1903, xxiv, 302.

The objects of laparotomy are: (1) to remove the focus of infection, such as a perforated appendix, or to prevent continued re-infection, as is done in suture of a perforated ulcer; and (2) to give exit to the inflammatory exudate and if necessary provide efficient drainage. The operative measures should be carried out rapidly, in order not to reduce the resistance of the peritoneum by exposure and drying, and the parts should be disturbed as little as is practicable with the attainment of the objects of the operation, so as to avoid spreading the infective microorganisms more widely in the peritoneal cavity. In former times removal of the intestines from the abdomen (evisceration) in order to clean them was attended by a mortality but little below that of cases not operated upon. To be successful the operation should be performed as early as possible after perforation of a hollow viscus or the onset of fulminating appendicitis, provided the patient be not in a condition of profound collapse. If this be present, operation must be delayed for some hours until, by stimulation, warmth, and other means, a reaction has been obtained. The main object of operative interference, therefore, is to remove the cause of peritonitis and so to prevent continued infection. Impressed with the high mortality of operation in established diffuse peritonitis, some surgeons, Ochsner especially, have adopted a conservative treatment, with the object of getting the disease localized and then operating. The treatment consists in keeping the intestines at rest, washing out the stomach, withholding food by the mouth, and giving nutrient enemas and plenty of water per rectum and by saline infusion.

As to the advisability of washing out the peritoneum, considerable diversity of opinion has existed. Formerly irrigation was almost universally employed, but it has now been largely given up and dry local sponging used instead. Dudgeon and Sargent have lately considered this question from the bacteriological standpoint, and conclude that in peritonitis due to *Streptococcus pyogenes* most thorough washing is desirable, as there is little risk of spreading the infection, and the phagocytes being powerless against this virulent infection, no harm is done by removing them. In peritonitis due to infection with *Bacillus coli*, on the other hand, irrigation is harmful, as it tends to spread the microorganisms widely over the peritoneal cavity, thus favoring absorption by the lymphatics of the diaphragm, and removing the protective exudation of phagocytes. In mild affections, such as those due to gastric perforation and intraperitoneal hemorrhage, the peritoneum should be most thoroughly cleansed so as to remove any fragment of food or clot.

In order to prevent absorption of bacteria by the lymphatics of the diaphragm, the pelvis is lowered and the patient placed in the semi-sitting (Fowler's) position, at an angle of 35 to 40 degrees, both before operation and for some days afterward.

When the intestines are greatly dilated from paralysis, the danger of fatal toxæmia from absorption of the foul contents of the bowel may be met by emptying the intestines. This can be done at the time of the operation by incising the bowel and expressing its contents, or, in more urgent cases, by inserting a Paul's tube into the intestine and leaving it for some hours in situ. Other methods have been employed, such as injecting purgatives, such as magnesium sulphate, by a syringe directly into the intestine during the operation (McCosh¹), after removing some of its contents (Mayland²);

¹ *Annals of Surgery*, xxvi, 691.

² *British Medical Journal*, 1899, i, 842.

by hypodermic injections of physostigmine under the skin; by the administration of enemata, methods often quite unsuccessful; or by the administration of calomel in one grain doses every hour until the bowels act; the calomel probably does good by its antiseptic action, as well as by leading to evacuation of the intestinal contents. Lawson Tait, who first advocated this line of treatment, gave saline purgatives. The purgative treatment of peritonitis has, however, met with considerable opposition both in the past and at the present time; it is useless when the intestines are paralyzed, and should only be adopted after operation and removal of the infecting focus. If employed before operation, it does harm by spreading the infection.

Transfusion.—In order to meet the urgent need for fluid and to combat collapse and toxæmia, enemata of saline solution should be given; the introduction of saline solution either intravenously or subcutaneously has also been extensively employed. Of the last two the subcutaneous method is the more convenient and can be frequently repeated if needed. The solution should be of the strength of a dram of salt to the pint of water (0.6 per cent.) and should be introduced by gravitation, the fluid in the reservoir being kept at 115° F. In this way as much as 15 pints can be infused in the twenty-four hours (Barnard). By filling the vascular system, absorption of poisons from the peritoneal cavity is inhibited, the current of the stream between the peritoneal cavity and the circulation being reversed, and the toxins already in the blood are diluted and their excretion by the kidney is favored. Continuous administration of fluid per rectum has been employed with good results.

Medical Treatment.—The patient must, it need hardly be said, be kept in bed; the shoulders should be raised with pillows, so that the exudation will gravitate toward the pelvis rather than toward the diaphragm, for in this way absorption of microorganisms into the circulation is minimized. If tenderness is extreme the bed-clothes may be kept off the abdomen by means of a cradle. The abdomen should be manipulated as little as possible, both because of the tenderness and because infection may thus be spread. The mouth should be frequently cleaned and kept moist.

The administration of purgatives must be prohibited, for by promoting peristalsis they would increase the pain and spread the infection more widely in the peritoneal cavity.

No food or fluid should be given by the mouth, and the urgent thirst should be relieved by enemata of saline solution. In the later stages the administration of sterilized olive oil hypodermically has been advised. Vomiting may be treated by washing out the stomach. It is doubtful whether bismuth, hydrocyanic acid, and drugs that calm an irritable stomach in other diseases are worth giving. Tympanites is difficult to relieve; enemata containing turpentine may be tried; or a long rectal tube may be passed every three hours and left in situ for half an hour.

The administration of opium in acute peritonitis has given rise to a good deal of discussion, but it may safely be said that when given now it is for humanitarian reasons, and not with the expectation of any permanent benefit. The reasons for giving opium or morphine are the severity of the pain, and also, it was formerly urged, to keep the intestines at rest and so prevent further spread of the infective process throughout the abdomen. It is also given when the condition of the patient appears hopeless, so as to relieve pain and suffering.

The objections to opium are weighty. It, of course, relieves and masks the symptoms in a marvellous manner, and so should never be given before a diagnosis has been made. But after a diagnosis has been arrived at, and especially when it is thought that the patient is too ill to stand operation, opium is not uncommonly given. Even in these conditions opium is undesirable, for since it induces intestinal paralysis and inhibits phagocytosis (Dudgeon and Sargent¹), it may just turn the scale against recovery.

Various local applications to the surface of the abdomen have been employed to relieve pain, such as hot and cold fomentations, the ice-bag, turpentine stupes, but often the slight pressure they exert gives rise to pain, and it is doubtful how much good is done in this way. Blistering the surface of the abdomen should not be countenanced.

Collapse may be treated by the external application of warmth, bandaging the limbs, and the hypodermic injection of strychnine. Adrenalin on a priori grounds should be an ideal remedy, as it acts locally on the nerve endings and does not stimulate the exhausted vasomotor centre. It has not fulfilled these anticipations; its effects are so transient that continuous intravenous injection of a solution of 1 in 50,000 to 100,000 with the addition of atropine (Crile) is the only method that has given good results (Hoddick²), and in some cases death has suddenly followed; it is known that adrenalin may induce pulmonary oedema, and Pearce³ has shown that experimentally it may cause rapid death from myocardial changes.

Serum Treatment.—It is to this that our hopes of future success in the treatment of acute peritonitis are directed. Since most of the fatal cases of peritonitis are due to a virulent infection with *B. coli*, it appears that a polyvalent anticolon serum is what is required to combat the disease; but as yet little advance has been made in this direction. In 25 cases of peritonitis, due to appendicitis, treated by injections of the serum of a horse immunized against thirty-one strains of *Bacillus coli*, recovery occurred in 9 or 36 per cent., and improvement was produced in all the others (Makins and Sargent⁴).

Polyvalent antistreptococcal serum has been tried in numerous cases, especially in puerperal septicaemia, unfortunately in the majority of cases without success.

ACUTE CIRCUMSCRIBED PERITONITIS.

Acute circumscribed or localized peritonitis may be conveniently divided into (1) non-suppurative and (2) suppurative.

Non-suppurative acute circumscribed peritonitis is extremely frequent and is due to extension of inflammation to the peritoneum. This is usually from organs in the abdominal cavity covered by peritoneum, such as the vermiform appendix, the intestines, the female genital organs, the gall-bladder, and liver; but acute inflammation of the pleura or pericardium may spread through the diaphragm to the surfaces of the spleen and liver; the results of this process are comparatively often seen in adhesions found after death on both sides of the diaphragm. A good example is that pro-

¹ *The Bacteriology of Peritonitis*, 1905, p. 188.

² *Zentralbl. f. Chir.*, Leipsic, 1907, xxxiv, 1193.

³ *Journal of Experimental Medicine*, 1906, viii, 400.

⁴ *Transactions of the Clinical Society of London*, 1907, xl, 146.

duced by an infarct in the spleen. There is pain on breathing like that of pleurisy, local tenderness, and from organization of the localized exudate adhesions frequently follow.

The chief clinical manifestation of acute circumscribed peritonitis is pain, which may vary in intensity but is confined to one area, and is made worse on pressure. A friction rub may be audible over solid organs, such as the spleen or liver. In rather infrequent cases acute circumscribed peritonitis may, by producing local paralysis of a coil of intestine, set up acute obstruction. Mayo Robson¹ has described this as the result of acute cholecystitis, and Halsted² has recorded dynamic dilatation of the first part of the duodenum and pylorus due to the same cause.

The main importance, however, of sharply circumscribed acute peritonitis lies not so much in its immediate effects as in results which it produces by the formation of local adhesions. By enclosing the affected area and shutting it off from the general peritoneal cavity, rupture of a perforating ulcer or of an abscess into the general peritoneal cavity may be prevented. On the other hand, the evil effects of a past attack of acute circumscribed peritonitis are shown by the occurrence of internal hernia due to strangulation of the intestine by bands formed from adhesions, and these around the stomach may give rise to persistent "adhesion dyspepsia," or to obstruction of the pylorus; some patients, usually women, suffer much from peritoneal adhesions in other parts of the abdomen, especially in the pelvis.

Treatment.—The treatment of acute circumscribed peritonitis is mainly symptomatic and directed to the relief of pain by external applications, such as hot fomentations or ice, the patient's feelings being consulted as to which is most effective. Opium or belladonna may be applied to the surface of the abdomen, or, if the pain be obstinate, leeches may be employed. The underlying conditions should, when possible, be treated.

Acute Pericolitis.—Just as appendicitis has become more frequent, so in recent years the manifestations of various forms of acute inflammation of the peritoneum covering the colon have become recognized. Pericolitis occurs in a number of conditions, and necessarily forms part of diffuse peritonitis and of various intraperitoneal abscesses and localized inflammations. As a descriptive heading, however, acute pericolitis should be confined to inflammation of the serous coat due to causes arising in the colon itself. Windscheid³ first described cases under the name of pericolitis in 1889.

Etiology.—Inflammation of the serous coat of the colon may be due to causes which allow microorganisms to traverse the walls of the colon, such as acute inflammation, ulceration, injury, or the passage of sharp foreign bodies through the walls of the colon. In cases of pericolitis there is often a history of previous constipation, but constipation is so common and pericolitis so comparatively infrequent that it appears that some further factor is necessary. This additional factor is probably stercoral ulceration, especially in acquired or false diverticula of the colon. The writer has recorded several examples of this condition.⁴ A closely allied condition has been described by Rixford,⁵ in which inflammation of the appen-

¹ *Medical-Chirurgical Transactions*, London, 1895, lxxviii, 117.

² *Johns Hopkins Hospital Bulletin*, 1900, xi, 1.

³ *Deutsch. Arch. f. klin. Med.*, Leipzig, 1889, xlv, 233.

⁴ *Lancet*, 1905, 1, 854.

⁵ *California State Journal of Medicine*, October, 1904, p. 296.

dices epiploicæ is due to infection conveyed from the colon. It has been suggested that pericolitis is rheumatic in origin (de Havilland Hall¹), but so far without any convincing evidence. As stercoral ulcers and false diverticula are more often present in the sigmoid flexure and descending colon, pericolitis is commoner in these parts of the large intestine. It is spoken of as perisigmoiditis and pericolitis sinistra, but it may occur in the transverse or ascending colon, especially at the flexures, where, from organization of adhesions (chronic pericolitis), the bowel may become narrowed, fixed, or kinked. Acute pericolitis may resolve, become chronic, or go on to the formation of a pericolic abscess.

Symptoms.—The symptoms of acute pericolitis are pain, loss of appetite, occasionally vomiting, some elevation of temperature, and quickened pulse. There is constipation, which, indeed, is often concerned in the causation, and also an absence of the manifestations of colitis, such as loose stools and mucus. The skin over the area may be hyperæsthetic, there is local muscular rigidity and deep tenderness, and in some instances an elongated tumor can be felt. Leukocytosis and indicanuria have been noted in some cases (Bittorf²). The clinical manifestations, therefore, run into those of coprostasis or faecal accumulation with an acute exacerbation. The symptoms, apart from their position, are very much like those of appendicitis. Cases on the left side, or pericolitis sinistra, can be readily recognized, but on the right side the diagnosis from appendicitis is necessarily extremely difficult, and can hardly be regarded as established before operation has shown that the appendix is healthy. The treatment consists in rest in bed, soothing applications to the abdomen, and the relief of constipation first by repeated enemas and subsequently by laxatives. The diet should be light and composed of food leaving little residue.

Epiploitis.—Epiploitis, or inflammation of the great omentum, is of course common in intraperitoneal inflammations on account of the tendency of the omentum to become attached to any focus of infection. Thus, inflammation of the omentum often accompanies appendicitis. Epiploitis has been especially described as a result of abdominal operations in which silk ligatures have been applied to the omentum. Schnitzler³ collected 28 cases after herniotomy, and it has also been met with two years after appendicectomy (Walther⁴). In some cases the condition has appeared long after the operation, there being a latent period of years. Cobb⁵ has described a case of inflammation of the great omentum due to a needle which appeared to have entered from the umbilicus. The condition of the omentum varies; it may be thickened and rolled into a firm mass showing inflammatory changes, and it may be adherent to other structures, or free. In a woman under my observation two radical cures for hernia had been done in 1899 and 1900; in 1903 the appendix was removed, and about two months later epiploitis occurred; a definite firm abdominal tumor appeared on the left side and a hectic temperature developed, but on laparotomy there was no abscess formation; part of the omentum was rolled up into a round mass, which microscopically showed developing connective tissue and some diminution in the number of fat cells. The inflammatory process may go on to the formation of an abscess.

¹ *Transactions of the Medical Society of London*, 1905, xxviii, 234.

² *Munch. med. Woch.*, 1904, li, 147.

³ *Wien. klin. Rundsch.*, 1900, xiv, 4.

⁴ *Bull. et mém. Soc. de chir. de Paris*, 1905, xxxi, 275.

⁵ *Boston Medical and Surgical Journal*, 1907, clvii, 18.

Symptoms.—The symptoms usually come on gradually, with fever, pain, constipation, and the presence of definite resistance or a tumor in the abdomen. The tumor may be lumpy, firm, adherent to the abdominal wall, tender, and imitate a new-growth. It may be chronic and eventually undergo resolution, but from contraction of adhesions the intestines may become kinked or otherwise obstructed. Epiploitis of this nature is rare, but if the conditions after which it occurs be borne in mind, the diagnosis should not be difficult.

Treatment.—Treatment in the early stage, when there is inflammation but not necessarily an abscess in the omentum, consists of soothing local applications. If fever persist and it appear probable that suppuration has occurred, laparotomy is the only course open.

There are transitional forms between non-suppurative and suppurative acute circumscribed peritonitis. It is not very rare in appendicitis to see a tumor-like swelling, which gradually disappears in a few days without any evidence that pus has been discharged into the bowel. The swelling is probably due to adherent intestines, with some inflammatory exudate enclosed between the coils of the bowel. In other cases, which at first show exactly the same features, an abscess eventually forms. In the same way inflammation of the omentum may be simple (epiploitis) or go to the formation of pus.

OMENTAL ABSCESSSES.

Omental abscesses are rare. As has been mentioned above, suppuration may supervene after epiploitis. Thus, several abscesses have been found in the omentum more than a year after an operation.

SUPPURATION IN THE MESENTERY.

Suppuration in the mesentery is usually secondary to disease of the intestine. As the result of infection conveyed from the small intestine, a mesenteric gland may suppurate, or pyogenetic infection may be engrafted on an old tuberculous adenitis. In cases of suppurative inflammation of the mesenteric veins there may be pus between the layers of the mesentery. Suppuration may also spread from the appendix into the mesentery. Barling¹ has published a series of cases, and has summarized the clinical aspect of the tumor; it is usually close to the umbilicus, mainly to the left side; when of considerable size it is practically fixed, but when of small size the swelling may not be palpable or if it can be detected is movable. The abscess may burst into the peritoneal cavity and set up general peritonitis.

PERIGASTRIC ABSCESS.

By a perigastric abscess is meant one that is due to infection from the stomach but does not come in contact with the under surface of the diaphragm, and, strictly speaking, is therefore not included in the more striking category of subphrenic abscess. It is true that suppuration due to disease of the pancreas, gall-bladder, or liver may be in contact with the stomach, and so

¹ *Clinical Journal*, London, 1907, xxx, 62.

perigastric in position, but these abscesses should not be described as perigastric.

Etiology.—In exceptional instances a simple perigastric abscess may be due to a minute perforation in the stomach wall, such as is produced by a sharp foreign body, or to slight leakage from a gastric ulcer, but, generally speaking, perforation of a gastric ulcer is followed by a gaseous subphrenic abscess. A perigastric abscess occurs in from 3 to 5 per cent. of all cases of gastric carcinoma (Fenwick¹), and is a late event. It is more often met with, so far as personal experience goes, in connection with rapidly growing and necrotic growths near the cardiac end; suppuration may then invade the left lobe of the liver. An abscess may form between the greater curvature of the stomach and the colon and then open into the large bowel, or may arise between the abdominal wall and the anterior surface of the stomach, and even point anteriorly or track down to the umbilicus. When a growth on the posterior wall of the stomach perforates, an abscess may form in the lesser sac of the peritoneum. The pus may be offensive, and if the abscess be opened during life food may be discharged.

Signs and Symptoms.—A superficial perigastric abscess may be palpable as a definite swelling, and even give rise to a projection and to reddening of the skin. But usually, especially in cases of gastric carcinoma, a perigastric abscess gives little in the way of physical signs; any increased resistance may be indistinguishable from that of the growth, and as a raised temperature is met with in 50 per cent. of the cases of gastric carcinoma (Osler and McCrae²), it is no proof of perigastric abscess, which, as already stated, occurs in a small percentage (3 to 5 only) of the cases.

Diagnosis.—It is practically impossible to diagnose between a perigastric abscess and the circumscribed form of phlegmonous gastritis, which is the same as an abscess in the walls of the stomach. A rapidly growing retroperitoneal sarcoma or hypernephroma may form a tender swelling in the left hyperchondrium and imitate a perigastric abscess. Thus in a girl, aged twenty-two years, sudden pain led to the discovery of a tender swelling below the left costal arch, which, from the raised temperature, was thought to be an abscess. Exploration proved that it was a necrotic hypernephroma. The sudden access of pain was probably due to hemorrhage into the growth.

PERICOLIC ABSCESES.

Abscesses due to infection from the bowel may occur in the immediate neighborhood of the large intestine from a number of causes: (1) From ulceration of the mucous membrane of the bowel, due to dysentery, accumulation of feces (stercoral), or, more rarely, to other factors, and by perforation by foreign bodies, circumscribed peritonitis may be set up, and sometimes a localized abscess may result. The abscess may be intraperitoneal, and may open into the bowel (exogenous ulceration) or into the peritoneal cavity, or may be retroperitoneal. (2) The mucous membrane of false diverticula of the colon, which are more often found in connection with the sigmoid flexure, may, as the result of the accumulation of fecal material inside them, undergo ulceration; perforation of these diverticula may lead to an abscess in the immediate neighborhood. (3) Suppuration may occur in connection with a carcinoma of the large intestine. This may

¹ *Cancer and Tumors of the Stomach*, 1902, p. 46.

² *Cancer of Stomach*, 1900, p. 46.

depend on perforation of the growth itself, on the passage of microorganisms through the floor of stercoral ulcers above the malignant stricture, or on perforation of these ulcers. The resulting tumor may be of considerable size, and on the right side must be differentiated from appendicitis with much inflammatory swelling, actinomycosis, and hyperplastic tuberculosis of the cæcum. An abscess formed in connection with a malignant stricture of the bowel may extend and open into the urinary bladder or even externally; rupture into the peritoneal cavity, however, appears to be infrequent. (4) Small but sharp foreign bodies present in food may penetrate the wall of the colon and, as Bland-Sutton¹ has shown, set up a faecal abscess in an appendix epiploica. This accident is more likely to occur in fat persons, for in thin individuals the foreign body more readily escapes into the peritoneal cavity.

Symptoms.—The symptoms are abdominal pain, tenderness, resistance, local prominence or distention, and perhaps a tumor, usually on the left side and suggesting left-sided appendicitis or even a new-growth. Constipation is commonly present, and also, not infrequently, vomiting. The abscess may increase in size and may rupture into the peritoneal cavity; the acute onset of peritonitis following on a period of pain and constipation may then suggest perforation above a malignant growth of the colon. Power² says that in some instances the abscess may become stationary, and from thickening of its walls give rise, for a time at least, to few clinical manifestations. The abscess may rupture into the intestine (Mayor³), or even into the urinary bladder, or externally (Patel⁴).

Diagnosis.—The diagnosis of these pericolic abscesses may be difficult, as from their comparative rarity some commoner condition, such as appendicitis in an unusual position, faecal accumulation, or malignant disease of the colon, may be suspected.

Treatment.—The treatment is purely surgical.

RESIDUAL PERITONEAL ABSCESES.

Residual peritoneal abscesses may occur in patients who have recovered from more or less general acute peritonitis. These abscesses may be found in any part of the abdomen, but are most often seen in the loins, pelvis, over the right lobe of the liver, or around the spleen. In tuberculous and pneumococcal peritonitis, and in suppuration in connection with the gall-bladder, abscesses may point at the umbilicus.

RETROPERITONEAL ABSCESES.

Suppuration behind the peritoneum may be due to a large number of causes, and when starting in one spot may spread to a considerable distance along the fascial planes by the lymphatics. A retroperitoneal abscess may be in contact with the diaphragm (vide Subphrenic Abscess). A psoas or iliac abscess due to tuberculous disease of the spine is, of course, retroperitoneal, but belongs to a somewhat different category, as does an iliac abscess due to calculous or tuberculous disease of the kidneys. Retro-

¹ *Lancet*, 1903, ii, 1148.

² *British Medical Journal*, 1906, ii, 1171.

³ *Rev. méd. de la Suisse Rom.*, Genève, 1893, xiii, 421.

⁴ *Rev. de Chir.*, Paris, 1907, xxxvi, 420, 698.

peritoneal suppuration may also be set up by disease of the bones of the pelvis or of the ribs. An abscess sometimes follows injury, especially in the neighborhood of the kidneys, and may be caused by minute rupture of viscera, or follow a hæmatoma. In other instances infection conveyed from the intestines to the retroperitoneal glands may result in the formation of an abscess. Perforation of viscera in situations where they are uncovered by peritoneum may be followed by retroperitoneal suppuration. Thus traumatic rupture, a penetrating bullet wound, or perforation of a stercoral ulcer in the colon may lead to a pericolic abscess, and an abscess around a carcinomatous stricture of the large bowel has been known to track into the thigh. Viscera entirely surrounded by peritoneum may, after contracting adhesions, perforate into the retroperitoneal tissues and set up an abscess; this is most commonly seen in connection with the vermiform appendix, but it also occurs in disease of the female genital organs. Retroperitoneal suppuration may be set up by infection introduced at operations, *e. g.*, herniotomy. These abscesses may rupture into the bowel or into other hollow viscera, such as the bladder or vagina.

Clinical Picture.—The constitutional manifestations—fever, rigors, loss of flesh, rapid pulse, and leukocytosis—are more constant than the local. There is often abdominal pain or uneasiness, while some deep tenderness on pressure may be present, and in some cases a palpable tumor. Local signs may, however, be absent, and the condition may be thought to be typhoid fever, generalized tuberculosis, or some obscure septicæmia. In some instances a diagnosis of an abnormally situated appendicular abscess may appear the most probable.

Treatment.—The treatment is purely surgical.

SUBPHRENIC ABSCESS.

Any abscess in contact with the under surface of the diaphragm, except when in the liver or in the spleen—the latter being a rare condition—is spoken of as a subphrenic abscess. From a clinical standpoint there are two forms of subphrenic abscess: (1) without any contained air—simple subphrenic abscess; and (2) containing gas—the subphrenic pyopneumothorax. Their clinical aspects are so different that separate descriptions are necessary, but it should be borne in mind that their anatomy is the same, the differences between them depending on the nature of the contents, and that the same cause may, in different cases, give rise to either of these forms of abscess. Thus an appendicular subphrenic abscess, although generally simple, may contain gas, and an abscess due to gastric ulcer, although usually gaseous, is not so in all instances. Nothnagel states that about half the subphrenic abscesses contain gas. In 59 cases of subphrenic abscess at St. George's Hospital, tabulated by Wahby, 32 were gaseous and 27 non-gaseous.

Subphrenic Abscess without any Gaseous Contents.—**Etiology.**—The simple subdiaphragmatic abscesses which do not contain gas may in the first instance be due to pyogenetic infection derived from a solid abdominal viscus in the neighborhood of the diaphragm; but suppuration may track up from the vermiform appendix or from the pelvis, *e. g.*, from a pyosalpinx. A simple subphrenic abscess may in rare instances arise in

connection with ulceration, simple or malignant, of the stomach or colon, but, as a rule, the resulting abscess in cases of perforating gastric ulcer contains air.

The causes of simple subphrenic abscesses may be conveniently tabulated in the order of their frequency:

1. Appendicitis is the most frequent cause of a simple subphrenic abscess. The resulting abscess is nearly always, as would be expected, on the right side, but a left-sided abscess may follow. The formation of a subphrenic abscess is a grave complication or sequel of appendicitis; thus among 86 fatal cases of acute appendicitis, subphrenic abscess occurred in 7, or 8.13 per cent. (Christian and Lehr¹). In some cases of subphrenic abscess the causal appendicitis may remain latent and be quite unsuspected. Appendicitis may give rise to a subphrenic abscess in several different ways: (a) An appendix which is retrocaecal, or runs up behind and to the outer side of the ascending colon and over the right kidney, may almost touch the right lobe of the liver; an appendicular abscess in this position (*i. e.*, the right kidney pouch) has a very short distance to travel to reach the under surface of the diaphragm. (b) An abscess around the appendix may spread upward in the paracolic groove to the right kidney-pouch and extend upward over the liver and under the diaphragm; this is the intraperitoneal route. The abscess may be retrocolic or retroperitoneal, and track upward. An appendicular abscess situated in the pelvis may extend upward along the paracolic grooves on both the right and the left sides of the abdomen, and so give rise to bilateral subphrenic abscesses. (c) A subphrenic abscess may be secondary to pyelphlebitis set up by appendicitis; such a pyelphlebitic abscess on the convexity of the liver may rupture and produce an abscess between the liver and diaphragm. (d) A residual abscess, the result of widespread peritonitis due to appendicitis, may form over the right lobe of the liver or around the spleen.

2. Suppuration in the liver very frequently sets up a non-gaseous subphrenic abscess. Collections of pus in the substance of the liver, such as tropical abscess, a suppurating hydatid cyst, multiple abscesses due to pyelphlebitis or to cholangitis, whether associated with gallstones or not, may extend to the convexity and set up an abscess between the surface of the organ and the diaphragm, or between the layers of the coronary ligament. The manipulations involved in operations on the liver or bile ducts may give rise to leakage of infective material from inflamed areas in the liver. As rare causes of a subphrenic abscess, attention may be called to leakage of a tuberculous or of an actinomycotic abscess near the surface of the liver, or even of a softened and infected gumma in that situation.

3. An abscess in the spleen is rare; it may be due to an infective embolus, pyæmia, follow typhoid fever and injury, or form part of suppurative pyelphlebitis. Suppuration in and around the spleen may in rare instances be due to an ulcer of the stomach or colon becoming adherent to and perforating into the substance of the organ. An abscess of the spleen, by leaking or rupture, readily sets up a subphrenic abscess. Traumatism, by giving rise to hemorrhage from or around the spleen, may be the first step in the production of a subphrenic abscess on the left side.

4. Although a localized abscess following perforation of a gastric ulcer

¹ *Medical News*, New York, 1903, lxxxii, 147

nearly always contains gas, a very minute perforation, such as that produced by a pin, may set up a simple abscess. It is possible that microorganisms may pass through the floor of an ulcer, and that suppuration may occur without any gross perforation. Among 27 non-gaseous subphrenic abscesses at St. George's Hospital, 2 were associated with a non-perforated gastric ulcer. A non-gaseous abscess occurs in about 3 per cent. of all cases of gastric carcinoma, but the resulting abscess is usually not in actual contact with the under surface of the diaphragm, and therefore rather comes within the category of perigastric abscess. The writer has seen a subphrenic abscess in connection with breaking down carcinomatous lymphatic glands close to the cardiac orifice of the stomach, the primary growth being in the lower end of the œsophagus.

5. Perforation of a duodenal ulcer usually floods the peritoneal cavity and sets up general peritonitis, but there are two ways in which it may set up a localized abscess (Maynard Smith¹): (a) Perforation of the serous surface of the duodenum may be followed by the escape of a moderate amount of fluid into the right kidney pouch, and by the formation of an extensive abscess. (b) Perforation of the duodenum behind the peritoneum may give rise to a localized retroperitoneal abscess. Osler estimates that 6 per cent. of subphrenic abscesses are due to duodenal ulcer. In his digest of 184 cases of duodenal ulcer, Cullen² finds that in 10 per cent. an abscess forms outside the duodenum, and that ultimately the ulcer opens into the abscess.

6. A pericolic abscess may, but often does not, lie in contact with the diaphragm. Pericolic abscesses at the hepatic or splenic flexures of the colon will necessarily be those most likely to become subphrenic.

7. Suppuration in or around the pancreas may spread so as to lie immediately under the diaphragm. The causes of pancreatic and peripancreatic abscess are numerous, such as pancreatitis, cholelithiasis, traumatism, perforation of an ulcer on the posterior wall of the stomach, and are dealt with elsewhere.

8. Suppuration in connection with the Fallopian tubes may, like an appendicular abscess in the pelvis, track up along the colon and give rise to a subphrenic abscess. In a case in St. George's Hospital actinomycosis of the Fallopian tubes was followed some months after removal by intra-hepatic and subphrenic abscesses.

9. Retroperitoneal suppuration starting from the kidneys, and from tuberculous disease of the spine or even of the ribs, must be mentioned, since the abscess may be subphrenic in position, but the clinical picture is usually different from that of the other forms of simple subphrenic abscess.

10. It is sometimes stated that an empyema, other forms of intrathoracic suppuration, or even pneumonia, may lead to a subphrenic abscess. It is, of course, true that tuberculous osteitis of the dorsal spine is commonly followed by a psoas abscess, but instances of subphrenic abscess undoubtedly due to intrathoracic causes are extremely rare; in 448 cases obtained by combining the statistics collected by Maydl, Grüneisen, and Perutz, there were 18 in which a subphrenic abscess was due to intrathoracic infection (Archibald³). In any given instance, therefore, it is more probable that the subphrenic abscess was the primary condition, but remained latent or

¹ *Lancet*, 1906, i, 895.

² *Scottish Medical and Surgical Journal*, Edinburgh, 1897, i, 643.

³ *British Medical Journal*, 1906, i, 1148.

was undetected for a time. It is, however, quite possible that exploration of an empyema may convey infection into the subphrenic space and so start an abscess below the diaphragm. In very rare cases suppuration in the posterior mediastinum, due to cesophageal ulceration, may track down and produce a subphrenic abscess. The infrequency with which infection spreads from the thorax to the abdomen is in marked contrast to the ease with which infection passes by the lymphatics from the peritoneum to the pleura; thus in 62 cases of streptococcal empyema, 14 spread from the abdomen (Pitt¹).

11. In some instances the focus from which a subphrenic abscess arose cannot be found. Possibly a minute traumatic rupture of a solid organ allowing hemorrhage to occur, or perforation of some part of the alimentary tract by a small but sharp foreign body may be the real cause in some cases. Thus traumatism may appear as the only antecedent event in the history. In 27 non-gaseous subphrenic abscesses at St. George's Hospital the average age was thirty-five years, being thirty-eight in 14 males and thirty-one in 13 females; in this series the incidence in the sexes was practically equal.

Morbid Anatomy.—Since a simple subphrenic abscess is in the majority of cases due to disease of the appendix or liver, it is much more often met with on the right than on the left side. It may be intraperitoneal or extraperitoneal, but is more commonly intraperitoneal. In 73 cases of subphrenic abscesses due to appendicitis collected by Elsberg,² 35, or 48 per cent., were intraperitoneal; 20, or 27 per cent., extraperitoneal; and in 18, or 25 per cent., the position was not determined. Retroperitoneal abscesses due to lesions of the spine or kidney are more allied to lumbar than to subphrenic abscesses. From a pathological point of view the greater cavity of the peritoneum is divisible into two parts: (a) Above the transverse mesocolon and omentum, supra-omental or supramesocolic; and (b) below the transverse mesocolon and omentum, or infra-omental. Intraperitoneal subphrenic abscesses occur in the supra-omental division of the greater sac. The anatomical boundaries of subphrenic abscesses vary according to the position of the abscess, and are further modified by the presence of old adhesions. The following descriptions may be regarded as applying both to simple and to gas-containing subphrenic abscesses.

The anatomical boundaries of subphrenic abscesses have been recently described by Box and Eccles,³ and Carnot.⁴ Barnard⁵ describes six forms of subphrenic abscess corresponding to six areas on the under surface of the diaphragm, four intraperitoneal and two extraperitoneal; the four intraperitoneal forms of subphrenic abscess are named (1) right anterior, (2) right posterior, (3) left anterior, and (4) left posterior; the areas on the diaphragm corresponding to these are defined and separated off from each other by the cruciform arrangement of the peritoneal ligaments—the falciform, coronary, and right and left lateral—of the liver. The two extra-hepatic subphrenic abscesses are (5) the right and (6) the left.

1. The right anterior subphrenic abscess is between the diaphragm and the convexity of the liver, and is bounded to the left by the falciform ligament, behind by the right lateral ligament of the liver, and in front by adhesions which vary somewhat in position; toward the right it communicates, unless shut off by adhesions, with the subhepatic or right kidney

¹ *Clinical Journal*, London, 1907, xxxi, 81.

² *Annals of Surgery*, 1901, xxxiv, 729.

³ *Clinical Applied Anatomy*, 1906.

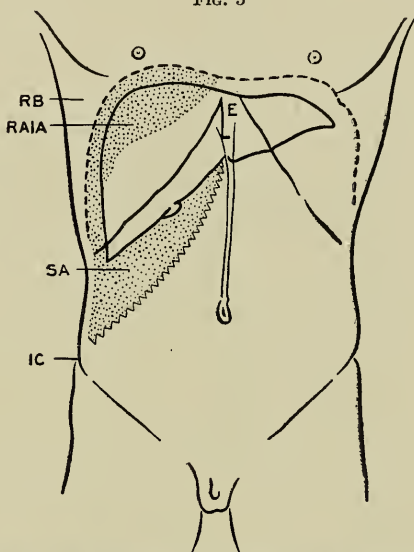
⁴ *Sem. méd.*, Paris, 1906, xxvi, 85.

⁵ *British Medical Journal*, 1908, i, 371.

pouch (Rutherford Morison), in which the right posterior intraperitoneal subphrenic abscess is situated.

2. This right kidney pouch may contain a pint of fluid before its contents run over into the remainder of the peritoneal cavity. Its boundaries are the right lobe of the liver above and in front, the hepatic flexure of the colon below, the parietal peritoneum of the lumbar region externally, the descending duodenum and lumbar spine internally, and the right kidney posteriorly. An abscess in this position commonly tracks up over the convexity of the liver into the right anterior subphrenic intraperitoneal space, and it may pass through the foramen of Winslow into the lesser sac of the peritoneum.

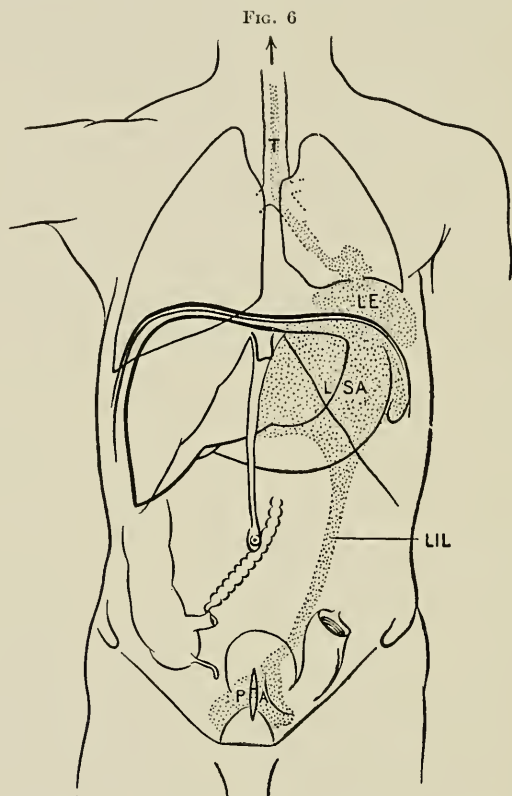
FIG. 5



The abdominal signs when a right anterior intraperitoneal subphrenic abscess (R.A.I.A.) is combined with a subhepatic abscess (S.A.) in the right kidney pouch as often occurs in appendicular cases. The former (R.A.I.A.) produces signs of compression and inflammation at the base of the right lung (R.B.). The latter (S.A.) produces a triangular swelling below the liver margin, limited toward the abdomen by a band of tender rigid adhesions running from the ensiform cartilage (E) back into the right loin, usually as low as the iliac crest, 1 C. Pus or gas may be found in contact with the abdominal wall within the triangle. This and the two following figures are reproduced, by his kind permission, from Mr. Barnard's paper in the *British Medical Journal*, 1908, i, 371.

3. The left anterior intraperitoneal abscess (Barnard) is in the dome of the stomach chamber and is in relation with the stomach and the spleen, or perigastric and perisplenic, and occupies a pouch which corresponds to the right kidney pouch (Box and Eccles) (Fig. 6). The phrenicocolic peritoneal ligament, binding the splenic flexure of the colon to the diaphragm and forming the suspensory ligament of the spleen, constitutes the lower margin of this pouch and, so to speak, dams up fluid in this peritoneal recess. The boundaries of left-sided intraperitoneal subphrenic abscesses are subject to some variation and Barnard's description is not followed here. Usually the abscess is between the left lobe of the liver, the diaphragm, the cardiac end

of the stomach, and the spleen; the abscess passes deeply toward the back of the abdomen in relation to the spleen, and has the left kidney, adrenal, and tail of the pancreas behind. The spleen may lie in the middle of the abscess, and the abscess is often tortuous and irregular in outline as it passes around the margins of the liver and spleen. In some cases the abscess is mainly to the left of the spleen, between it and the diaphragm; according to Hunt¹ this position of the abscess follows perforation of an ulcer on the posterior



Left anterior intraperitoneal subphrenic abscess (L.S.A.) due to infection conveyed from pelvic appendicular abscess (P.A.) along (L.I.L.) the left internal lumbar fossa (Barnard), or left internal paracolic groove (Jenkin and Maynard Smith). It has set up left-sided empyema (L.E.), which has burst into the trachea. (Barnard.)

wall of the stomach. The abscess may also occupy the space between the diaphragm and the left lobe of the liver, passing downward and to the right, under the liver, and in front of the stomach. Such an abscess is usually the result of suppuration in the left lobe of the liver or in connection with the anterior surface of the stomach.

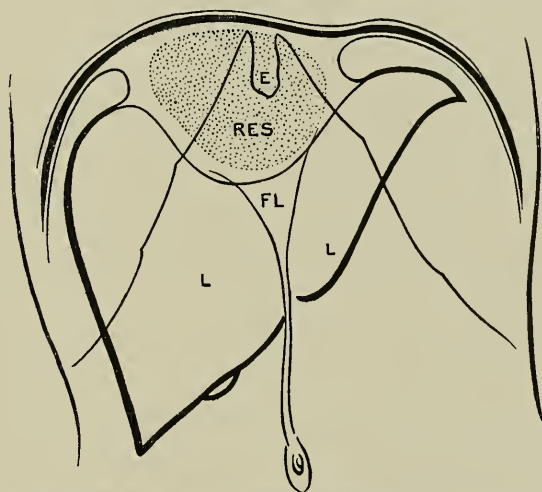
4. An abscess confined to the lesser sac of the peritoneum is rare; it constitutes Barnard's left posterior intraperitoneal abscess.

¹ *Transactions of the Medical Society, London, 1905, xxviii, 75.*

The median boundary of abscesses in contact with the under surface of the diaphragm is formed by the falciform ligament of the liver, which may be displaced by the pressure of the abscess, but is hardly ever perforated. It is extremely rare to find suppuration on both sides of the falciform ligament at the same time; but suppuration starting in the pelvis may track up in the paracolic grooves on both sides of the abdomen, and so give rise to abscesses in contact with the hepatic and splenic flexures of the colon.

5. The right extrahepatic abscess is in the uncovered space between the layers of the coronary ligament of the liver, and is closely allied to, if not the same as, the suprahepatic abscess described by Cantlie.¹ It is nearly always due to intrahepatic suppuration.

FIG. 7



The signs of a right extraperitoneal subphrenic abscess (*R.E.S.*), pointing in the epigastrium below the ensiform process (*E.*) and having separated the layers of the falciform ligament (*F.L.*) so that the abscess may be opened extraperitoneally. Note that the liver is pushed down in this case, and freely movable. (Barnard.)

6. The left extraperitoneal abscess (Barnard) is near the left kidney and is rare. Barnard finds that the right anterior and left anterior intra-peritoneal abscesses, due in the main to appendicitis and to perforated gastric ulcer respectively, are the commonest; next in order of frequency comes the right extraperitoneal abscess.

The walls of the abscess are formed of shaggy fibrin, the diaphragm, surface of the liver, intestines, and so forth being infiltrated for a considerable distance with organizing granulation tissue. The size of the abscess varies very considerably in different cases; it may be as small as a man's fist or larger than a child's head, and contain several pints of pus. In cases of appendicular origin the pus is often putrid.

A subphrenic abscess readily sets up inflammation of the adjacent pleura,

¹ *British Medical Journal*, 1899, ii, 646.

and leads to an effusion which may be serous and sterile, fibrinous, or purulent. This occurs in about half the cases of subphrenic abscess. Like an abscess in the liver, it may perforate the diaphragm and open into the pleura, leading to empyema, into the lung, thus setting up gangrenous pneumonia, multiple abscesses, and extensive disorganization of the lower lobe, or in very rare cases it may open into the pericardium. In Laug's¹ 173 cases of subphrenic abscess the diaphragm was perforated in 67, and in Martinet's² 138 cases in 33; both these series include gaseous as well as non-gaseous abscesses. Intraperitoneal abscesses are less prone than extraperitoneal abscesses to perforate the diaphragm. The pus in the empyema may be extremely offensive; Dieulafoy³ has reported cases in which a simple or non-gaseous subphrenic abscess of appendicular origin gave rise to a pyopneumothorax, the gas being due to bacterial activity and not to a communication with the lung or any of the hollow viscera. As the result of the rupture into the lung of a subphrenic abscess, set up by intrahepatic suppuration, a bronchobiliary fistula, with copious bile-stained expectoration, may be established. Rupture externally or into the hollow viscera—the stomach or colon—is rare. Inflammation may extend to the portal vein and set up adhesive or, as a later stage, suppurative pyelephlebitis. Rupture into the peritoneal cavity is a very grave event and is usually fatal. It occurred once only among 23 cases of spontaneous rupture of a subphrenic abscess (Barnard).

Bacteriology.—The colon bacillus is commonly present; streptococci, staphylococci, *Bacillus pyocyaneus*, and *B. typhosus* have also been found. In Archibald's case, which was secondary to an empyema, both abscess cavities contained pneumococci. Actinomyces have also been detected in the pus of a subphrenic abscess.

Symptoms.—These are somewhat obscure and vary to some extent according to the origin and the position of the abscess. Thus an abscess between the convexity of the liver and the diaphragm will resemble an intrahepatic abscess, while a purulent collection in the lesser sac of the peritoneum (bursa omentalis) will present features like those of a pancreatic cyst. In fact, subphrenic abscesses in these two positions are, from a clinical point of view, closely bound up with diseases of the liver and pancreas respectively.

The onset of symptoms is usually gradual, and it may be difficult to distinguish between those due to the primary disease, such as appendicitis or calculous cholangitis, and those depending on pus formation under the diaphragm. Thus after an operation for an appendix abscess, continued fever, diarrhoea, and quick pulse may persist, and after some time signs at the base of the right lung suggest that an abscess has formed under the diaphragm. In some instances the thoracic manifestations, such as pleurisy, are the first to attract attention. This gradual onset contrasts with the sudden pain which marks perforation of the stomach in the majority of the cases of subphrenic pyopneumothorax. In some instances there is a sudden onset with a rigor.

Physical signs may be absent or so slight as to be easily overlooked, and are less obvious than in gaseous subphrenic abscess. From the point of view of physical signs, simple subphrenic abscesses have been divided into two groups: (1) those in which the thorax is mainly affected, and (2) those in which the signs are mainly abdominal.

¹ Thesis, Moscow, 1895.

² Thèse de Paris, 1898.

³ Cliniques médicales de l'Hôtel-Dieu de Paris, 1901-02, iv, 105.

1. Since a subphrenic abscess is usually nearer the spinal attachment of the diaphragm than the epigastrium, the signs are chiefly found posteriorly, and are more thoracic than abdominal. There is dulness at the base of one, usually the right, lung, which slowly increases upward toward the inferior angle of the scapula; there may be bronchial breathing from compression of the lung, a friction rub from concomitant pleurisy, or, from pleural effusion, absence of breath sounds. The physical signs, therefore, resemble those of pleural effusion. Examination with the *x*-rays may be of the greatest use, by showing that the diaphragm on the affected side is pushed upward by a dark area, and that the excursions of the diaphragm are restricted or absent.

2. In some cases, in which the manifestations are abdominal, a subphrenic abscess may pass forward, present anteriorly below the costal margin, and give rise to tenderness, fulness, and prominence of the hypo-chondrium and epigastrium, restricted movement of the ribs, muscular resistance, and a more or less definitely palpable tumor, which is dull on percussion. The dulness is constant and does not alter when the patient's position is changed. Under an anæsthetic the muscular rigidity passes off, and the intestine may pass in front of the abscess cavity and so lead to resonance where there was previously dulness on percussion (Godlee¹). As the abscess approaches the surface the skin may become œdematous and red, and fluctuation may be obtained; the manipulation necessary to elicit this sign is, however, not without risk of producing rupture into the general peritoneal cavity. Pain is usually not a severe symptom, and, as a rule, is pleuritic and transient; it may be felt at the angle of the scapula. The temperature is nearly always raised and may show the oscillations of suppuration, but in a few instances fever is absent. The pulse is quick, the respirations are often irregular, painful, and rapid, especially when the thorax is much invaded; cough is exceptional. Leukocytosis up to 30,000 may be present. The appetite is poor, wasting is considerable, and there may be frequent perspirations, especially at night, and considerable secondary anæmia. Clubbing of the fingers never occurs, and when it is present in a case thought to be one of subphrenic abscess the lesion will probably turn out to be above the diaphragm (Acland²). With continued temperature and progressive loss of strength the patient steadily goes down hill unless the abscess be freely drained. The abscess may extend and open into the pleura, lung, bronchus, into the peritoneum, one of the abdominal viscera, or even externally through the ribs or at the umbilicus. In exceptional instances the abscess has burst into the pericardium, or has tracked up into the neck (Cullen). Pulmonary embolism may result from thrombosis of branches of the inferior vena cava. The writer has seen an infarct of the liver in association with portal thrombosis in a case of subphrenic abscess. Death usually results from exhaustion. The duration of the disease, if it be left to run its natural course, is somewhat prolonged—from two to three months.

Diagnosis.—When there is no antecedent condition, such as appendicitis or suppuration elsewhere in the abdomen, the existence of a subphrenic abscess may not be suspected unless there be a palpable swelling. On the other hand, when after operation on an appendix abscess, or on the gall-

¹ *Lancet*, 1905, i, 482.

² *System of Medicine* (Allbutt and Rolleston), 1907, vol. iii, p. 1007.

bladder or biliary passages, the patient's general condition still suggests suppuration, careful examination of the bases of the lungs by percussion, auscultation, and the *x*-rays may show signs compatible with a small abscess or effusion. The affected area should then, as Barnard directs, be systematically explored with a syringe, the needle of which is at least three inches long and sufficiently large to enable thick pus to be withdrawn. The patient is fully prepared for operation and anaesthetized, and the needle is passed deeply—for three inches—into the 10th intercostal space in the scapular line, and then, if no pus be found, into the 9th, 8th, 7th, and 6th spaces in turn; if necessary, the same spaces in the midaxillary line are explored from below upward. With this thorough and systematic method of exploration Barnard has not had any failures in detecting an abscess. In the cases in which a subphrenic abscess presents under the anterior abdominal wall the exploring syringe should not be used.

Differential Diagnosis.—The chief difficulty in distinguishing between subphrenic abscess and other intra-abdominal conditions on the right side is in deciding whether the collection of pus is inside the right lobe of the liver or between it and the diaphragm. Enlargement of the liver downward is in favor of intrahepatic suppuration, but in many instances it is impossible to come to a positive conclusion, and, indeed, a subphrenic abscess is often due to leakage of an intrahepatic focus of suppuration. A subphrenic abscess on the left side presenting as a palpable swelling must be distinguished from soft tumors, innocent, such as perirenal lipomas, or malignant, such as retroperitoneal sarcomas. The raised temperature and signs of septic absorption should be taken into account in forming an opinion, but a swelling in the left hypochondrium of rather sudden onset, accompanied by pain and a raised temperature, has turned out to be a retroperitoneal sarcoma. In some instances a large, false abdominal aneurism on the left side does not pulsate and no bruit is audible over it; the diagnosis may then be very difficult, as is shown by cases reported in a paper by the late Mr. T. Holmes:¹ "On Pulsating Tumors which are not Aneurismal and on Aneurisms which are not Pulsating Tumors." The presence of persistent leukocytosis is in favor of abscess, but a posthemorrhagic leukocytosis may occur in aneurism.

The main difficulty, however, is not in distinguishing between subphrenic abscess and other abdominal conditions, but between subphrenic abscess on the one hand, and an empyema or pleural effusion on the other. It has happened that when an abscess diagnosed as an empyema is drained, particles of food are found in the discharge, and for the first time indicate the underlying cause. In a simple subphrenic abscess on the right side the liver is more depressed, the breath sounds less cut off at the base of the lung behind, and the constitutional symptoms are more marked in comparison with the thoracic dulness than in an empyema. Examination with *x*-rays will be of assistance in showing that the diaphragm is pushed up instead of being depressed.

The question whether the collection is above or below the diaphragm is often rendered more difficult by the not infrequent presence of a pleural effusion secondary to the suppurative process under the diaphragm. When an empyema and a subphrenic abscess are both present the existence of the latter is extremely likely to be overlooked, and its detection is very difficult

¹ *St. George's Hospital Reports*, 1875, vii, 173.

indeed. This combination, if suspected, can be recognized by the withdrawal by the exploring syringe of pus from one interspace and serous fluid from one above it. This sign, however, is not infallible, for in rare cases a loculated pleural effusion may contain serum in one loculus and pus in another.

Brief reference may be made to two signs which have been described in order to decide whether a given collection of fluid is above or below the diaphragm. If the aspirating needle is introduced into a cavity above the diaphragm no movement of the needle occurs on respiration, if the needle passes through the diaphragm the extrathoracic part of the needle moves upward in inspiration and downward in expiration; this is Fürbringer's sign, and when present is regarded by Nothnagel as pathognomonic. The second sign is Pfuhr's; when the needle enters a subphrenic abscess the manometric pressure and the outflow of pus are greater during inspiration and less during expiration, while the reverse holds good in the case of an empyema. These two signs depend on the normal excursions of the diaphragm being maintained, but this does not always hold good; for example, a pleural effusion may paralyze the diaphragm and so lead to mimicry of the condition in a subphrenic abscess. Further, it must be borne in mind that puncture of the diaphragm may lead to the spread of infection from the abdomen to the thorax, or vice versa, and is therefore not devoid of danger.

Prognosis.—This in non-gaseous subphrenic abscess depends on the condition being recognized and promptly treated by exploration and thorough drainage. If the abscess be not opened the case will almost certainly terminate fatally; further, it is most important that the abscess should be opened before it has extended into the lung or given rise to septicæmia. Even when opened there is the risk that some loculus of the abscess may not drain properly. The difficulty in diagnosing and, when the existence of the abscess is suspected, in accurately locating the abscess makes the prognosis grave. A subphrenic abscess may be opened successfully, but an empyema may be overlooked and subsequently prove fatal. As already pointed out, the diagnosis of a simple subphrenic abscess is more difficult than that of a gaseous subphrenic abscess.

Most of the available statistics as to the mortality of subphrenic abscess contain both simple and gas-containing abscesses, so that it is difficult to compare the mortality of the two forms. In Elsberg's 73 cases of subphrenic abscess (15 per cent. of which were gaseous) due to appendicitis, 51 were operated upon, with recovery in 40, or 78.4 per cent., while of 22 not operated upon 18, or 82 per cent., died. Out of Barnard's 76 cases, 36, or 47.4 per cent., died; 12 cases were only recognized after death and were not operated upon. Of the remaining 64 the mortality was 37.5 per cent., but after making allowance for imperfect diagnosis and treatment, Barnard concludes that the mortality should be 16 per cent.

Treatment.—That of simple subphrenic abscess is purely surgical, and consists in opening and draining the abscess cavity.

Subphrenic Pyopneumothorax.—The peculiarity of this form of subphrenic abscess is that at the bedside it imitates an ordinary pneumothorax or pyopneumothorax, such as most commonly results from rupture of a tuberculous vomica in the lung. The existence of a gaseous abscess underneath the diaphragm was first diagnosed during life, as a logical

deduction from the physical signs, by G. H. Barlow¹ as far back as 1845, but although the condition was described by subsequent observers, such as Bouchard,² Hilton Fagge,³ Eisenlohr,⁴ and Cossy,⁵ it was not until Leyden's⁶ article, in 1880, that it became generally recognized and the subject of numerous papers, such as Maydl's monograph in 1894. It seems that since perforated gastric ulcers have been treated by immediate operation this form of subphrenic abscess has become less common.

Etiology.—The causes of a gaseous subphrenic abscess are less numerous than those of a simple subphrenic abscess. Perforation of a gastric ulcer is the most important cause of a gaseous subphrenic abscess. In the sixty years, 1846 to 1906, there were about 26,000 autopsies at St. George's Hospital, among which Wahby found 32 gaseous subphrenic abscesses, 25 (21 females, 4 males) of which, or 77.4 per cent., were due to perforated gastric ulcer. In order that the resulting peritonitis should be localized, certain conditions are necessary; thus preëxisting adhesions which prevent the gastric contents from spreading widely over the peritoneum are of great importance. It is, however, highly probable that in certain circumstances perforation of the stomach may lead to a localized abscess even in the absence of these adhesions; the occurrence of perforation in a stomach containing but little food, in a patient in the prone position, so that only a small quantity of the gastric contents escapes, and does not travel far into the peritoneal cavity, and then mainly into the upper part, make up a combination of circumstances in which the inflammatory process may become circumscribed instead of generalized. The position of the perforation is another factor of importance: if on the posterior surface, a localized abscess is likely to follow, whilst if it is on the anterior surface, general infection of the peritoneum is apt to occur. Perforation of an ulcer close to the pylorus may lead to a localized abscess in the right kidney pouch. Perforation of the stomach due to carcinoma is a rare cause of a gaseous abscess.

Perforation of a duodenal ulcer usually gives rise to general peritonitis, but in a small minority of the cases a subphrenic pyopneumothorax on the right side results. As long ago as 1862, Bouchard recorded a case. Out of 32 gaseous subphrenic abscesses at St. George's Hospital, 6 (3 males, 3 females), or 19 per cent., were due to perforation of a duodenal ulcer.

An abscess in the neighborhood of the stomach or duodenum has in rare instances perforated (exogenous ulceration) into the alimentary canal, with the result that a gaseous subphrenic abscess is produced. A gas-containing abscess due to a purulent collection ulcerating into the colon was recorded by Vanlair.⁷ A suppurating hydatid cyst of the liver may rupture into the alimentary canal and become gaseous, but this condition, of which Dévé⁸ has collected 7 examples, is not the same as an ordinary subphrenic pyopneumothorax. A suppurating hydatid of the liver, from infection with anaërobic organisms, may contain gas, and in rare instances, by rupturing

¹ *London Medical Gazette*, 1845, i, 13.

² *Bull. Soc. anat.*, Paris, 1862, xxxvii, 309.

³ *Guy's Hospital Reports*, 1874, xix, 213.

⁴ *Berl. klin. Woch.*, 1877, xiv, 539.

⁵ *Arch. gén. de méd.*, Paris, 1879, exliv, 526.

⁶ *Zeit. f. klin. Med.*, 1880, i, 320.

⁷ *Rev. de méd.*, Paris, 1893, xiii, 561.

⁸ *Rev. de chir.*, Paris, 1907, xxxv, 529.

into the space between the liver and the diaphragm, may produce a subphrenic pyopneumothorax on the right or left side, according to the position of the cyst in the right or left lobe of the liver. In Maydl's¹ collection of 179 cases of subphrenic abscess there were 3 of gaseous subphrenic abscess due to hydatid cysts of the liver.

Perforation of an ulcer in the colon very seldom produces a gaseous abscess; Osler² refers to a subphrenic pyopneumothorax due to an ulcer of the splenic flexure of the colon.

An appendicular abscess, although usually simple, may be gaseous; this was so in 15 per cent. of the 73 cases of subphrenic abscess due to appendicular infection analyzed by Elsberg.³ A case of subphrenic pyopneumothorax recorded as long ago as 1877 was regarded as secondary to appendicitis (Eisenlohr). The presence of gas in these abscesses was at first considered to be due to secondary communication with some air-containing viscus, but it is, in many instances at least, of bacterial origin.

Morbid Anatomy.—The situation and boundaries of subphrenic abscesses have been described. In contrast to simple subphrenic abscesses, which are much commoner on the right side, gas-containing subphrenic abscesses are, in the great majority of cases, on the left side. This is due to the preponderating influence of perforated gastric ulcers as the causal factor.

The diaphragm is pushed up so as greatly to encroach on the cavity of the thorax. The walls of the abscess cavity are much thickened, lined by adherent and often necrotic fibrin, and infiltrated with organizing granulation tissue, the deeper layers of which show, in cases of some standing, well-formed cicatricial tissue and, in the case of muscles, infiltration and degeneration. The perforation in the stomach or duodenum is usually open, but in some instances becomes closed with fibrin. The size of the cavity may vary considerably; sometimes these abscesses are very large, and have been known to contain in addition to gas a quart of purulent fluid with an admixture of gastric contents of an extremely offensive character. The abscess may erode the substance of the spleen. Perforation into the general cavity of the peritoneum occurs in a few cases. The inflammatory process readily spreads through the diaphragm and sets up pleurisy with effusion and collapse of the lower lobe of the lung. The effusion is usually serous or sero-fibrinous, but may be purulent, and in this event the lung may be disorganized by multiple abscesses. Perforation of the diaphragm may occur and be followed by a true pyopneumothorax or, when the lower lobe of the lung is already adherent to the diaphragm, by suppurative destruction of the lung or gangrenous pneumonia; or the abscess may rupture into the bronchi. Pericarditis may be found, but is not so frequent as pericardial friction during life.

Age and Sex.—As most cases are due to perforation of a gastric ulcer, the patients are chiefly young women. In the 32 cases at St. George's Hospital the average age was thirty-three years, being thirty years in the 24 females and forty-two years in the 8 males. Vanlair recorded a gaseous subphrenic abscess in a boy aged six years.

Symptoms.—The onset is usually sudden, with very severe pain, due to perforation of the stomach; in this respect it differs from the simple sub-

¹ *Ueber subphrenische Abscesse*, 1894, Vienna.

² *Principles and Practice of Medicine*, 1905, p. 503, sixth edition.

³ *Annals of Surgery*, 1901, xxxiv, 729.

phrenic abscesses in which the onset is often insidious. In some cases, however, the leakage from the stomach is quite latent. The patient's history often shows past dyspepsia, pain after food, and other manifestations of gastric ulcer. The pain, which is agonizing and may be accompanied by collapse, is situated in the upper part of the abdomen, usually on the left side. The onset may be accompanied by vomiting, which may or may not persist afterward; the vomited matters may contain blood and bile. It is important to realize that vomiting may occur after perforation of a gastric ulcer, and that this event does not seriously militate against that accident. Directly after the onset the abdomen becomes extremely tender, so that accurate examination may be impossible at first; as a rule, it rapidly becomes distended, although in men it may be rigidly contracted, tympanitic, and does not move on respiration. The temperature becomes raised, the pulse and respiration rates are rapid, rigors may occur, leukocytosis is present, there is nocturnal sweating, and the patient has the general appearance of suppuration with abdominal pain and some respiratory embarrassment.

Physical Signs.—The physical signs may very closely resemble those of a true pneumothorax or pyopneumothorax. There is a tympanitic note on percussion over an area of distention in the upper part of the abdomen; the altered note extends up into the thorax for a considerable distance, but not to the extreme apex. On the right side the diaphragm may be displaced as high as the second rib, but the presence of the heart prevents the same amount of upward displacement on the left side. The tympanitic note may be mainly behind the sternum. There is dulness posteriorly on the affected side when the patient is lying on his back, and when turned on his side the positions of the dull and tympanitic areas alter as in a pyopneumothorax. A bell or coin percussion note, amphoric breathing, succussion, and metallic tinkling may be heard over the swelling and part of the chest. When the left side is affected the heart is displaced upward, but only slightly to the other side, thus differing from what occurs in a true pneumothorax. When the abscess is on the right side the liver may be greatly displaced downward and be palpable below the umbilicus; the hepatic dulness will then be absent from its normal position. The costal margin is pushed out and the lower intercostal spaces may bulge as in pneumothorax.

The occurrence of thoracic complications is almost constant. Signs of pleurisy rapidly come on, and there is some dulness with impaired entry of air at the base due to effusion, but in some instances the signs are those of pneumonia of the base of the lung on the corresponding side. The effusion is usually serous at first, but may subsequently become purulent. A pleuropericardial or pericardial friction rub is not uncommon.

Secondary parotitis occasionally occurs, and if suppuration supervenes may prove a very serious complication. In rare instances subcutaneous emphysema has occurred.

Diagnosis.—The common error is to regard as a pyopneumothorax a gaseous abscess which is below the diaphragm. While the history and mode of onset frequently point to the abdomen rather than to the thorax, the signs suggest a pyopneumothorax from which it can be most certainly distinguished by a skiagram showing the position of the diaphragm. But it must be borne in mind that distention of the colon or stomach may push the diaphragm up.

The physical signs in favor of a gaseous subphrenic abscess are that the

pneumothorax appears to be partial, not universal, as shown by the presence of normal breathing over the upper part and apex of the lung; that the heart's apex is displaced upward and but little to the opposite side; that a bell note and other signs of an air-containing cavity can be obtained well below the limits of the thoracic cavity for example below the left costal margin; and that cough and expectoration are late in appearing, or insignificant.

A subphrenic pyopneumothorax is often complicated by a pleural effusion—serous or purulent; the diagnosis of these two concomitant conditions may be greatly aided by the withdrawal of pus and gas from one interspace, and of serum or pus of a different character from a higher interspace. The fluid from a subphrenic abscess comes out more freely during inspiration, while in a pleural effusion its exit is accelerated during expiration. It must be remembered, however, that puncture of the chest, although often desirable for purposes of determining whether, and if so where, operative procedures should be carried out, is not devoid of the danger of spreading infection from the pleura to the peritoneum, or vice versa.

The diagnosis from a pleural effusion rests on the detection of the signs of a cavity containing fluid and gas, on the changing dullness in a gaseous subphrenic abscess and on the absence of this change in a pleural effusion (Godlee), on the lateral displacement of the heart in a pleural effusion, and upon its upward displacement in a subphrenic gaseous abscess.

Abdominal distention due to flatulence may be accompanied by disappearance of the normal liver dullness and by a bell note, but the liver cannot be felt to be displaced downward.

A diaphragmatic hernia on the left side which contains the stomach gives rise to signs resembling in many respects those of a pyopneumothorax, but the abdomen falls in instead of being distended, and the heart is pushed over to the right side very much more than in a subphrenic pyopneumothorax. In making a diagnosis the *x*-rays should be employed, and a metal probe passed into the stomach and its position noted.

Prognosis.—In subphrenic pyopneumothorax this is always grave, partly because the extension of inflammation to the pleura and lung is a frequent and serious complication, and partly because the large abscess cavity may not close up and the patient dies from the effect of toxic absorption.

Treatment.—This is surgical, and consists in opening and draining the abscess cavity.

SIMPLE CHRONIC PERITONITIS.

This condition may for convenience be divided into (1) circumscribed or localized areas of chronic peritonitis, and (2) diffuse chronic peritonitis. There are necessarily transitional cases in which the chronic peritoneal change is, on the one hand, not sufficiently localized to be included in the first category, and, on the other hand, is far from implicating the whole of the peritoneal cavity. As a matter of fact, chronic peritonitis is comparatively seldom found to be universal and equally well-marked over the entire abdominal cavity. The change may be advanced in the upper part of the abdomen over the liver, spleen, and omentum, and comparatively slight in the pelvis.

Localized Chronic Peritonitis.—It is important to distinguish between (a) the adhesions that result from a past acute inflammation of the peritoneum and (b) the progressive or chronic inflammatory process which alone deserves the name of chronic peritonitis. The causes of localized chronic peritonitis are numerous, and may be enumerated in the order of frequency with which the various intra-abdominal organs are responsible.

The female genital organs very commonly give rise to chronic pelvic peritonitis which produces dense adhesions around the Fallopian tubes, ovaries, and uterus. On the right side the vermiform appendix and the broad ligament may become so matted together that even after death it is extremely difficult to determine in which of these structures the inflammatory process originated. The important factors in causing chronic perimetritis and pelvic peritonitis are gonococcal and puerperal infections. It is highly probable that hemorrhages from the Fallopian tubes may, as the result of subsequent microbic activity, be responsible for chronic pelvic peritonitis.

A chronic gastric or duodenal ulcer may produce progressive localized peritonitis in the immediate vicinity. This may result in firm adhesions. The contraction of the thickened peritoneum may play a part, with the cicatrization of the ulcer itself, in producing hour-glass contraction of the stomach, duodenal stenosis, or even stricture of the common bile duct and jaundice.

Appendicitis is an important cause of localized chronic peritonitis in the right iliac fossa. Chronic peritonitis may also be limited to the orifice of hernial sacs, to the surface of the intestines in chronic intussusception, and occasionally, it is said, to the flexures of the colon in chronic fecal accumulation.

Chronic cholecystitis, especially when associated with gallstones, may set up very marked local peritonitis which unites the gall-bladder to the neighboring viscera, especially the pyloric end of the stomach. In this event symptoms of pyloric obstruction are prone to occur.

In hydatid or other cysts, such as ovarian, there may be such thickening of the overlying peritoneum that the condition resembles a lamellar or corneal fibroma. As the result of friction or pressure, thickening of the capsule or chronic capsulitis of the spleen and liver may occur. Localized thickenings, white in color, raised above the surface of the capsule of the spleen, are common in splenomegaly; they are composed of dense, white, fibrous tissue arranged in parallel lines, with a few connective-tissue corpuscles, and are spoken of as lamellar or corneal fibromas. Much less commonly similar masses are found on the surface of the liver. A very common, although structurally slightly different, condition is the chronic peritonitis seen on the anterior surface of corset-livers and due to the pressure of the corset or of a belt.

Clinically, the results of localized chronic peritonitis are variable. In the case of the lamellar fibromas of the spleen there are no signs or symptoms. Adhesions and induration around the stomach, and flexures of the colon and elsewhere may induce symptoms due to interference with the natural movements of the viscera. Thus in the case of the stomach there may be persistent pain after food, or "adhesion dyspepsia;" this is not uncommon in women formerly the subjects of gastric ulcer, and may be thought to be due to a relapse of that disease. As a very rare result of perigastric chronic peritonitis, there may be compression of the common bile duct and jaundice.

In one example of this, secondary to a chronic ulcer near the pylorus, there was such extensive fibrosis that a tumor, regarded as malignant during life, could be felt. As already mentioned, chronic peritonitis around the gall-bladder may, by surrounding the pylorus, lead to pyloric obstruction and dilatation of the stomach; there may be so much thickening as to imitate slow-growing carcinoma of the pylorus.

Chronic peritonitis around the colon, especially the flexures, may give rise to constipation and pain, especially after defecation; according to the position of the lesion, the condition may imitate appendicitis or malignant disease of the colon. This subject of chronic pericolicitis has recently attracted considerable attention.

As the result of chronic pelvic peritonitis in women, there may be uterine disturbance and such persistent pain that the patients become chronic invalids. In one case examined after death the dense fibrosis, due to salpingitis, simulated malignant disease, and had so compressed the ureters as to lead to fatal uræmia.

Patients vary much in the way in which they feel such pain; some are martyrs to these painful peritoneal adhesions, while in others—probably in the great majority—there is little or no inconvenience. Like the adhesions left as a relic of a past attack of localized acute peritonitis, those of chronic peritonitis may produce acute intestinal obstruction by kinking and strangulation of the intestine. In some instances the persistent pain may justify laparotomy. Short of this, the treatment should be directed to diet, prevention of constipation, local measures for the relief of pain, and to improvement of the general health.

Chronic peritonitis involving the mesocolon of the sigmoid flexure (mesosigmoiditis) and the mesentery of the lower part of the ileum (chronic mesenteric peritonitis) has been especially described by Riedel as the result of constipation. It is not a common condition. There is a radiating thickening of mesocolon and the sigmoid flexure, which is an important factor in the production of volvulus of the sigmoid flexure; the thickening of its mesentery leads to considerable shortening in the length of the small intestine. The thickening begins in the mesentery and mesocolon away from the intestinal attachment, and, as Nothnagel points out, this militates against the view that the chronic peritoneal change is due to faecal accumulation; it has been suggested by Virchow and Nothnagel that it is the result of inflammation of the mesenteric and other glands secondary to intestinal infection in which constipation may play a part.

The clinical features of this special form of chronic peritonitis are somewhat difficult to interpret correctly. There may be kinking or volvulus of the sigmoid flexure, and it has been suggested that cases of intestinal obstruction that recover on medical treatment may be of this nature. In a case of this kind in St. George's Hospital the signs suggested carcinoma of the descending colon.

Chronic pericolicitis, a sequel of an acute attack of acute pericolicitis, may lead to very considerable thickening of the walls of the colon and may imitate malignant disease (D'Arcy Power,¹ Moynihan²). Massage has been recommended to relieve the condition.

¹ *British Medical Journal*, 1906, ii, 1171.

² *Transactions of the Clinical Society of London*, 1907, xl, 31.

Chronic hyperplastic tuberculosis of the intestine, which has attracted a considerable amount of attention of recent years, usually takes the sub-mucous form, but in a few instances, of which Kidd¹ has collected 6 examples, it is subserous and in at least 3 cases has attacked the sigmoid flexure; it is not improbable that some cases described as subserous fibromas were in reality of this nature.

Diffuse Chronic Peritonitis.—Under this heading it is convenient to consider cases which have in common a diffuse peritoneal inflammation of chronic and progressive character, not manifestly due to tuberculosis or new-growth. It is sometimes called chronic simple peritonitis to distinguish it from the two forms just mentioned. Chronic proliferative and chronic indurative peritonitis are synonyms sometimes employed, and since ascites is extremely common, the name chronic exudative peritonitis is almost as widely applicable. As will be seen below, the heading—Diffuse Chronic Peritonitis—describes an anatomical condition with more or less uniform clinical manifestations, but, like dysentery or colitis, it cannot be regarded as a single disease.

Etiology.—Chronic simple peritonitis may be due to one of a number of different factors, and it is convenient to divide the cases into three groups. There is considerable difference of opinion as to the relative importance of the causal factors. The classification of chronic peritonitis here adopted is: (1) When associated with varying degrees of chronic inflammation of the pericardium and pleuræ. (2) When associated with arteriosclerosis and granular kidneys. (3) When associated with other conditions. The first two categories are the most important.

1. Chronic simple peritonitis may be combined with chronic inflammatory changes in the pericardium and pleuræ of varying extent and intensity. The association of similar inflammatory changes, whether acute or chronic, in two or more serous cavities has been called polyserositis and polyorrhymenitis.² When the condition is chronic it has also been termed multiple progressive hyaloserositis or Concato's disease. The chronic inflammatory changes in the thorax that may be found in association with simple chronic peritonitis vary in extent and intensity; thus there may be chronic indurative mediastinopericarditis with obliteration of both pleuræ, chronic mediastinitis alone, or adherent pericardium. Cases associated with adherent pericardium are the most usual; Gilbert and Garnier³ described a group of 11 cases as "symphyse péricardio-périhépatique." In some cases of long standing the thickened and adherent pericardium may be calcified; the peritoneal change is then advanced and the liver may be cirrhotic, thus differing from its usual condition of that organ—chronic venous engorgement and fatty change—in chronic peritonitis. It is generally considered that when chronic intrathoracic and peritoneal serositis are associated, the primary focus is more often intrathoracic; but it must be remembered that infection spreads more rapidly from the abdomen to the thorax than vice versa.

The original starting point of the inflammation may thus be on either the upper or under surface of the diaphragm; when there is an adherent pericardium, especially if it be calcified, it is probable that the original

¹ *Lancet*, 1907, i, 9.

² *Vide* F. Taylor, *British Medical Journal*, 1900, ii, 1693.

³ *Bull. et. mém. Soc. biol.*, Paris, 1898, l, 48.

focus was in this position, but it is quite possible that pericarditis might be secondary to peritoneal infections. The original lesion may be an acute pericarditis or perihepatitis, or there may be recurrent attacks. But on whichever side it starts it spreads through the diaphragm, the constant movements of which interfere with the natural cure of the morbid process, and so induce the chronic inflammatory process which is most marked on each side of the diaphragm and fades off in the more distant parts of the adjacent serous membranes. The chronic peritonitis is, therefore, most advanced over the liver and spleen, and this may be so noticeable that the condition was graphically described by Curschmann¹ as *Zuckergussleber* (iced liver), and is sometimes spoken of as universal chronic perihepatitis; these names suggest a more intimate connection with the liver than really exists, since more careful examination would show that, although more prominent over the liver, the chronic peritoneal change is not confined to that region.

The pericardial pseudocirrhosis described by Pick² is probably allied to this form of chronic peritonitis; Pick, however, believed that a latent adherent pericardium gives rise to circulatory disturbance in the liver, hepatic fibrosis, ascites, and that, although as a result of the ascites and repeated tapplings, chronic peritonitis might be induced, it was more or less accidental and a secondary event. Nicholls³ and Kelly⁴ have drawn special attention to the association of chronic peritonitis with similar intra-thoracic changes.

2. The association with arteriosclerosis and granular (arteriosclerotic) kidneys was insisted on by Hale White,⁵ who found it in 19 out of 22 cases of universal perihepatitis and peritonitis, but some observers, especially Nicholls, do not attach much importance to its etiological influence. The relation between arteriosclerosis and the peritoneal change may be either (a) direct; it has been suggested that arteritis in the small vessels of the peritoneum sets up a chronic fibrotic change. Another hypothesis is that the uræmic poisons, circulating in the blood of patients with arteriosclerosis and the resulting renal disease, produce chronic inflammatory changes in the serous membranes; or (b) indirect; the diminished bactericidal power of the blood, shown by Flexner⁶ to be common in cases of chronic renal disease, may allow infection with organisms of no great virulence to take place.

3. Simple chronic peritonitis may be met with in combination with various intra-abdominal lesions, or its starting point may be quite obscure. The associated lesion, such as syphilitic disease of the liver, may reduce the resistance of the peritoneum, or, as in the case of chronic duodenal ulcer or cholecystitis, may provide a local focus for peritoneal infection. In such cases a widespread chronic inflammatory process results instead of the local reaction which would be expected; this may be compared with the formation of large cheloids after minute injuries, and possibly the excessive reaction may depend on a low resistance, inherent or acquired, of the peritoneum in that individual.

Alcoholism has been regarded as a factor, and would reduce the resistance

¹ *Deut. med. Woch.*, 1884, x, 564.

² *Ztschr. f. klin. Med.*, Berlin, 1896, xxix, 385.

³ *Studies from the Royal Victoria Hospital*, Montreal, 1902, i, No. 3.

⁴ *American Journal of the Medical Sciences*, Philadelphia, 1903, cxxv, 116.

⁵ *Transactions of the Clinical Society of London*, 1888, xxxi, 219.

⁶ *Journal of Experimental Medicine*, 1896, i, 559.

of the peritoneum and by setting up gastro-intestinal inflammation favor microbic infection; of the influence of other chemical poisons, such as lead, there is no evidence. The chronic venous engorgement due to heart and other obstructive diseases would act in much the same manner. The relation of ascites to the chronic peritonitis is usually considered to be that the chronic peritonitis causes the ascites, but it is conceivable that in some circumstances the reverse may hold good; thus, in chronic ascites due to backward pressure of heart disease, repeated tapplings may favor a low form of infection, which is not necessarily introduced from without, but may be carried by the blood stream. That tapping may lead to chronic inflammation is shown by examination of the cells found in ascitic effusions; an effusion which at the first tapping shows a preponderance of endothelial cells, and is therefore mechanical in origin, may in subsequent tapplings show an altered cell count due to superadded changes (Cade,¹ Gilbert and Villaret²). Chronic peritonitis set up in this way may be at first local and in the neighborhood of the puncture, and gradually become more widespread and possibly universal.

In some cases traumatism appears to be the exciting factor, and it is quite conceivable that by damaging the viscera it would allow of a low form of infection of the peritoneum. Lastly, no obvious cause may be forthcoming.

Pathogeny.—Since the process is evidently a chronic and progressive form of inflammation, it has naturally been surmised that it is the result of infection with microorganisms of a low grade of virulence. Nicholls suggests the *Bacillus tuberculosis*, *Bacillus coli*, and *Bacillus typhosus*. Louis (1825) regarded chronic peritonitis as always tuberculous; in modern times Picchini (1891) argues that subacute and chronic polyorrhymenitis is practically always tuberculous, and F. Taylor, who quotes him, accepts this in a modified way. The recognition in recent years of a special form of tuberculosis, the chronic hyperplastic, which is best known in connection with the ileocaecal part of the intestine, but has also been recognized in the ureter and female urethra (Hartmann³), in bursae, and elsewhere, suggests that many cases of chronic peritonitis are tuberculous; in other words, that there may be a chronic hyperplastic tuberculous peritonitis that chronic hyperplastic tuberculosis of the intestine does to ordinary intestinal tuberculosis. As bearing on this view, it may be pointed out that, by means of Jousset's method of inoscopy, tubercle bacilli are now found in many cases of ascites which do not otherwise suggest peritoneal tuberculosis, and that tuberculosis with fibrotic changes is a not uncommon complication of arteriosclerosis. The last point may help to explain the association of arteriosclerosis and granular kidney with simple chronic peritonitis.

Morbid Anatomy.—The appearances in simple chronic peritonitis vary considerably from the more marked changes of the "iced" organs to a general slight opacity of the peritoneum. In a well-marked case the peritoneum is covered by a thick membrane which resembles a thin layer of cartilage; it is dull white or glistening, and pearly in color, and may show pigmentation. This dense layer, especially over solid organs, such as the

¹ *Arch. de méd. expér. et d'anat. path.*, Paris, 1906, xviii, 769.

² *Compt. rend. Soc. biol.*, Paris, 1906, lx, 820.

³ *Mém. et bull. Soc. chir.*, Paris, 1906, and *Ann. gyn. et d'obstét.*, Paris, 1907, lxiv, 1.

liver and spleen, for which it forms a firm casing, may show a few depressions like the pitting of sand by rain-drops. It is possible that these deficiencies are the result of rupture of the dense fibrous tissue, induced by its own contraction. On the other hand the thick membrane may be locally accentuated in the form of fibrous elevations or nodules, perhaps more often seen on the parietal peritoneum; to this condition the name "*peritonitis fibrosa*" has been applied; these fibrotic elevations when small may closely resemble miliary tubercles, or when large, nodules of disseminated malignant disease.

The thick membrane, which may be even one-quarter of an inch in depth, can usually be peeled off the liver and spleen, leaving their capsules in a fairly normal condition. From its marked tendency to cicatricial contraction it squeezes the organs and leads to atrophy; in extreme instances these organs may therefore become so atrophied as to be isolated in the abdominal cavity and lose their normal relations. The omentum becomes shortened and rolled up into a firm cord, often in irregular knots, running transversely across the abdomen; in some instances hardly a trace of the omentum is left. In like manner the mesentery becomes contracted, so that the small intestines are anchored to the spine and cannot float in the ascitic fluid, "and, if a hernia had existed, it will sometimes be found to have been completely reduced" (Hodgkin¹). At the same time there may be such considerable diminution in both the length and diameter of the alimentary canal that the *valvulae conniventes* become very closely placed—Prof. W. H. Welch has shown me a specimen in which the longitudinal section of the intestine looked like a comb—and the colon may be no larger than the small intestine. This gives rise to impaired absorption. The condition has been called "*peritonitis deformans*." Subacute or acute peritonitis may supervene.

Adhesions are often present, but their extent varies greatly. They are more often seen over the solid and relatively fixed organs, since the peristalsis of the intestines interferes with their formation and may rupture them when formed. The remains of adhesions may be seen as filamentous or villous tags on the small intestine, and sometimes resemble miliary tubercles. Adhesions are not uncommon between the liver or spleen and the diaphragm, and may be dense or delicate. The presence of definite cords may give rise to acute strangulation of the intestine, but this is rare in this form of chronic peritonitis. When there are sufficient adhesions the ascitic fluid may become encysted, and when the adhesions are so numerous as to obliterate a large area of the peritoneal cavity fluid effusion may be slight in amount.

The liver and spleen are often described as "iced," or as showing universal chronic capsulitis, and in the case of the liver as "*Zuckergussleber*." This condition, also called universal chronic perihepatitis, was formerly regarded as a distinct morbid entity, and many cases of chronic ascites, in reality due to chronic peritonitis, were regarded as the result of chronic perihepatitis, the existence of the more widespread peritoneal change being ignored or regarded as a secondary result of the recurrent ascites and consequent tapplings. From the frequency of ascites in hepatic cirrhosis the liver was considered the important factor in the production of ascites, and disease of its capsule was therefore accepted as a sufficient cause of ascites without any reference to the peritoneal change. This was not unnatural, since in

¹ *Lectures on the Morbid Anatomy of the Serous and Mucous Membranes*, i, p. 152, 1836, London.

many cases the change is much more manifest over the liver than elsewhere. On the liver and spleen the thickened membrane is confined to the serous surfaces, and does not affect the areas between the reflections of peritoneum, such as the coronary ligament. The thickness of the membrane varies in different parts, it modifies the shape of the viscera, rounds off the edges, and covers up natural depressions. The gall-bladder may be almost buried, and is usually collapsed, but constriction of the bile duct or portal vein, contrary to what might naturally be expected, is very rare. In a case at one time under my care prevertebral chronic peritonitis leading to obstruction of the lymphatics above the level of the receptaculum chyli and so to elephantiasis was found at the autopsy (Bernstein and Price¹). The organs are compressed, and usually show chronic venous engorgement, but any considerable extension of fibrosis from the capsule into the substance of the organs is very rare.

Microscopically there is extremely dense fibrous tissue laid down in parallel lamellæ, with a few nuclei between. The fibrous tissue is prone to undergo hyaline change, hence the condition has been called hyaloseriosis (Nicholls); the structure of the fibrous tissue is the same as that of the so-called lamellar fibromas of the spleen, which, indeed, are localized foci of the same process. The deeper layers of the membrane show bloodvessels with collections of leukocytes and mast cells. Nicholls was unable to find any evidence of fibrin. As the wrinkled peritoneum can be seen underneath, the formation of the membrane is due to organization of exudation, and not to hyperplasia of the peritoneum.

Clinical Picture.—The condition most commonly occurs in middle life, but it may be seen in children; the sex incidence is about equal.

The cases may present different manifestations, but the characteristic result is chronic ascites, which requires numerous tapplings and recurs with remarkable persistence. The interval between successive tapplings may diminish in progressive cases so that paracentesis may become necessary every week; more than 100 tapplings may be required, and in Rumpf's cases the abdomen was tapped 301 times in sixteen years. After tapping, the rolled up omentum may be clearly felt running across the abdomen, and is often more distinctly palpable toward the left hypochondrium. It must not be regarded as the margin of an enlarged liver, or as the greater curvature of the stomach infiltrated with carcinoma. The other signs of ascites will be found. The abdomen when distended may be almost uniformly dull, since from retraction of the mesentery the small intestines are tethered to the spine and unable to reach the abdominal wall. In rare instances friction is audible through a stethoscope applied to the abdominal walls. In some cases the fluid is encysted and the abdomen is irregular, so that the diagnosis may be very difficult. Signs of concomitant disease, such as adherent pericardium, valvular disease of the heart, pleural effusion, or renal disease, may be present. The urine is somewhat scanty and may present the characteristics of granular kidney. Jaundice is very rare, and when present is due to some complication.

Symptoms.—The onset is, as a rule, gradual; but symptoms may date from a febrile attack, and are usually vague. The first thing noticed is swelling of the abdomen followed by abdominal uneasiness and a sense of

¹ *British Medical Journal*, 1907, i, 617.

fulness and weight, but pain is absent or slight and only brought on by movement. There is naturally some loss of strength, and the muscles are flabby, but emaciation is slow in appearing. Patients may feel well and except for the abdominal embarrassment, able to be up. Respiration may be somewhat hampered, constipation is usual and no doubt in part depends on the weakened state of the abdominal muscles, and there may be some flatulency and dyspepsia. Gastro-intestinal hemorrhage does not occur. In the later stages considerable wasting and œdema of the feet supervene. It is noteworthy that œdema of the feet occurs long after ascites has been well established.

The fluid varies to some extent in its characters, usually it is like serum, of a citron color, and of a specific gravity of 1015 or more. It may clot and form threads of fibrin or large flocculi. In some instances, in which subacute inflammation has supervened, it is turbid from the presence of cells. It may be milky or chyliform, and after tapping may become blood-stained.

Diagnosis.—This rests mainly on the presence of chronic and recurrent ascites, for which no other cause is forthcoming. It must be distinguished from other causes of ascites, such as tuberculous and malignant peritonitis and hepatic cirrhosis. It is probable that in the future cytological examination of the ascitic fluid will render this comparatively easy.

Prognosis.—This should be guarded; recovery is rare, but the course is slow, and as many as 100 tapplings may be required. Some cases of the recurrent ascites which are diagnosed as simple chronic peritonitis certainly recover after a number of tapplings. Nicholls found that the duration varied between two and sixteen years. The average duration of Ramsbottom's 9 cases was 624 days.¹ Death is generally due to some intercurrent infection, such as pneumonia, but in cases associated with adherent pericardium and chronic mediastinitis it may be due to cardiac failure.

Treatment.—Treatment should, in the first instance, be directed to the cause, such as renal disease, heart affections, syphilis; otherwise it is palliative and symptomatic. No permanent good can be anticipated from laparotomy and the attempt to produce artificial adhesions on the lines of the Talma-Morison operation for the relief of the ascites of portal cirrhosis of the liver. Paracentesis should be performed when required; often this gradually becomes more frequent and may be necessary every week. Injection of adrenalin chloride, one dram in an ounce of distilled water, into the peritoneal cavity before the cannula is withdrawn is worth a trial.

Diuretics, such as caffeine, may be employed from time to time, and tonics will usually be necessary. Good hygienic conditions and fresh air are desirable. It is not necessary for the patient to remain in bed in the earlier stages. Some relief to the feeling of weight in the distended abdomen may be obtained from the use of a binder or belt.

TUBERCULOSIS OF THE PERITONEUM AND TUBERCULOUS PERITONITIS.

The ordinary clinical heading of tuberculous peritonitis covers cases with somewhat different morbid lesions; thus, although tuberculous inflamma-

¹ *Medical Chronicle*, Manchester, 1906, xlv, 7.

tion confined to the peritoneum is, strictly speaking, all that is implied, it is customary to include cases which have in addition tuberculous ulcers of the intestine and tuberculosis of the intra-abdominal lymphatic glands, since these conditions are commonly combined and often cannot be separated during life.

The *incidence* of peritoneal infection in tuberculosis has been differently estimated. In 531 cases of pulmonary tuberculosis examined after death at the Brompton Hospital, there was tuberculous peritonitis in 22, or 4.1 per cent. (Fowler and Godlee¹). In 197 cases of tuberculosis at the Boston City Hospital the peritoneum was infected in 14, or 7.1 per cent. (Bottomley²). In 1393 cases of tuberculosis at Breslau there was tuberculous peritonitis in 226, or 16.2 per cent. (Borschke³). In 300 cases of fatal tuberculosis of various parts of the body at St. George's Hospital the peritoneum was affected in 56, or 18.6 per cent.

Etiology.—Age.—The disease may occur at any period of life, but it is rare in babies and in old age. Osler records a patient aged eighty-two years, while among 59 cases of tuberculosis in children tabulated by Colman⁴ the youngest case of fatal peritoneal tuberculosis was one and a half years. It is most frequent between twenty and forty years of age. The belief sometimes expressed, that the disease is commoner in children, is probably the result of confusion between chronic intestinal disturbance with a distended abdomen, formerly spoken of as “*tabes mesenterica*,” and true peritoneal tuberculosis. In 306 cases of tuberculous peritonitis under the age of fifteen, nearly half were between the ages of three and seven years, and the sex incidence was equal (Faludi⁵).

Sex.—There is a marked divergence between statistics obtained from postmortem records and those of operation as to the sex incidence. Post-mortem records show that it is more frequent in males; in König's 107 cases, 89 were males. In operation cases females are greatly in excess; according to Nothnagel, whose estimate is probably based on König's series of operation cases, 90 per cent. are in women; of Osler's⁶ 131 cases, 60, or 54 per cent., were women. The preponderance of female cases discovered at operation appears to depend on the frequency with which tuberculosis of the Fallopian tubes serves as the starting point for peritoneal tuberculosis.

Disposing Factors.—Cirrhosis of the liver shows a decided tendency to be complicated by peritoneal tuberculosis; thus in 584 cases of hepatic cirrhosis, obtained by combining the statistics of Lancereaux, Kelynnack, Yeld, Rolleston and Fenton, tuberculous peritonitis occurred in 53, or 9 per cent. No doubt concomitant or antecedent alcoholism favors the incidence of tuberculosis generally, but the catarrhal condition of the intestine and the portal obstruction probably play a part in the localization of the infection. Injury has, in some cases, preceded the onset of symptoms. Pregnancy, according to H. Kelly,⁷ has a definite causal relation to tuberculous peritonitis; in 28 per cent. of his cases the illness was dated from childbirth or a miscarriage. Heredity would naturally be expected to be of some impor-

¹ *Diseases of the Lungs*, p. 358, London, 1898.

² *Medical and Surgical Report*, Boston City Hospital, 1900, p. 118.

³ *Virchow's Archiv*, 1892, cxxvii, 121.

⁴ *British Medical Journal*, 1893, ii, 740.

⁶ *Johns Hopkins Hosp. Rep.*, 1891, ii, 67.

⁵ *Jahr. f. Kinderh.*, 1905, lxii, 304.

⁷ *Operative Gynecology*, 1906, ii, 237.

tance in the incidence of tuberculous peritonitis, but in 25 cases investigated by Bottomley there was a history of tuberculosis in 2 only.

Bacteriology.—Peritoneal tuberculosis has been regarded by Raw¹ as due to infection with bacilli of bovine tuberculosis absorbed from the alimentary canal and introduced with the food. He considers that pulmonary tuberculosis and the secondary intestinal ulceration which occurs as the result of swallowing sputum in at least 50 per cent. of fatal cases of chronic pulmonary tuberculosis, are due to infection with the bacillus of human tuberculosis.

Tubercle bacilli are comparatively seldom found in the peritoneal effusion by ordinary microscopic examination. Jousset's method of inoscopy is, however, more successful in leading to their detection. This comparative scarcity of bacilli in the fluid is also seen in tuberculous pleural effusion, and is thought to depend on adhesion of the bacilli to the serous membrane. Injection of the ascitic fluid into guinea-pigs shows that bacilli are present in the effusion.

Paths of Infection.—The miliary tubercles seen on the peritoneum in some cases of generalized tuberculosis are due to hæmic infection, the bacilli reaching the peritoneum by the bloodvessels, but in these cases the peritoneum merely shares with the rest of the body in the universal outburst of tubercles and seldom or never shows any accompanying inflammation. Tuberculous infection usually reaches the peritoneum through the lymphatics. The bacilli may be conveyed from the intestine and vermiform appendix, the lymphatic glands, the pleuræ, the suprarenals, prostate, vesiculæ seminales, and the testes.

According to Nothnagel, the lungs are primarily affected, without any intestinal lesions, in the majority of cases of peritoneal tuberculosis; but in Borschke's 226 cases of tuberculous peritonitis there was primary pulmonary tuberculosis with marked intestinal ulceration in 140 cases, or 62 per cent. It is, however, remarkable how often tuberculous ulceration of the intestine secondary to pulmonary tuberculosis is seen without diffuse tuberculous peritonitis. Thus among 382 fatal cases of pulmonary tuberculosis at the Brompton Hospital, London, tuberculous ulcers were present in the intestine in 296, or 77.4 per cent., while tuberculous peritonitis occurred in 15 only, or 4 per cent. (Fowler and Godlee). The occurrence of tuberculous peritonitis secondary to primary intestinal tuberculosis is variously estimated. German statistics are to the effect that primary intestinal tuberculosis even in children is very rare, and it is therefore not surprising that among Borschke's 226 cases of tuberculous peritonitis not a single one was secondary to primary intestinal tuberculosis. British statistics give a percentage of primary intestinal tuberculosis in children ranging up to 28.1 per cent. of the cases of tuberculosis (Shennan²) and averaging 19 per cent. as against 4 per cent. in Germany and 3 per cent. in the United States (Harbitz³). In women tuberculous disease of the Fallopian tubes is a prolific source of tuberculous peritonitis. Primary tuberculosis of the peritoneum, in which there is no tuberculous focus elsewhere in the body, and in which the path by which bacilli have got into the peritoneum is not obvious, is rare. Borschke's series of 226 cases contained only 2.

¹ *British Medical Journal*, 1906, ii, 357.

² *Edinburgh Hospital Report*, 1900, vi, 130.

³ *Journal of Infectious Diseases*, Chicago, 1905, ii, 189.

Morbid Anatomy.—Before describing the morbid appearances of diffuse tuberculous peritonitis a few words may be devoted to localized tuberculosis of the peritoneum. Local tuberculosis is always present in the subserous tissues of tuberculous intestinal ulcers, and occasionally gives rise to localized peritonitis, as shown by tags of adhesions. There may be localized tuberculosis in the neighborhood of the vermiform appendix or Fallopian tubes. In some instances a hernial sac when operated upon has been found to show localized tuberculosis. In generalized miliary tuberculosis the peritoneum may share in the infection and present a number of small miliary tubercles without any obvious inflammatory reaction.

In tuberculous peritonitis the morbid appearances vary very considerably. Strictly speaking, distinct types of tuberculous peritonitis from a pathological point of view do not exist, for they overlap and one described form may be only the later stages of another. While bearing in mind that in any classification the different groups must merge into one another, it is convenient to recognize distinct forms of such a variable disease. It may be advisable to quote some of the groups of tuberculous peritonitis described by various authors. Murphy¹ gives the following forms: (1) Disseminated miliary with ascites; (2) nodular, ulcerative, or perforative; (3) adhesive, obliterative, or cystic; and (4) suppurative, circumscribed or general, mixed infection. Osler describes (1) acute miliary with serofibrinous or bloody exudation; (2) chronic tuberculosis, with larger growths which tend to caseate and ulcerate; the exudation is purulent or seropurulent and is often sacculated, and (3) chronic fibroid tuberculosis; the tubercles are hard and pigmented and there is little or no exudation, adhesions being present. Allchin² adopts a somewhat similar arrangement of (1) miliary tuberculosis, acute or subacute, (2) ulcerous, and (3) fibrous forms of the disease. Moynihan³ gives the following forms: (1) ascitic; (2) fibrous, with cases showing a transition to the previous form, namely, presenting pseudocysts, and (3) suppurative.

The following method of dividing the anatomical lesions in tuberculous peritonitis depends on the amount of adhesions present; the cases are divided into (1) the ascitic; (2) the loculated, including (*a*) suppurative, those called chronic tuberculous by Osler, and ulcerous by others, and (*b*) cases of encysted ascites; and (3) obliterative, corresponding to the groups called chronic fibroid tuberculosis by Osler, fibrous or adhesive by others.

1. In the ascitic form there are few or no adhesions, and as a result the exudation is free in the peritoneum. There is miliary tuberculosis which may be acute, subacute, or chronic. In the more acute forms congestion and inflammation of the peritoneum are engrafted on to acute miliary tuberculosis. The small miliary tubercles vary in size from that of a millet seed downward, and are scattered over the parietal and visceral layers of the peritoneum, mesenteries, and omentum, and are often more numerous over the diaphragm. In the chronic forms the tubercles are larger, are surrounded by fibrosis, and are often somewhat pigmented; the peritoneum is thickened and the naked-eye appearances may exactly imitate those of malignant disease. The omentum becomes rolled up into a hard mass, the

¹ *American Journal of Obstetrics and Diseases of Women and Children*, 1903, xlviii, 1904, xlix.

² *System of Medicine* (Allbutt and Rolleston), 1907, iii, p. 958.

³ *Abdominal Operations*, 1906, 111.

mesentery is shortened from cicatricial contraction so that the intestines become tethered to the spinal column, and from a similar process the intestine becomes shortened in length. These changes resemble those in simple chronic peritonitis.

In rare instances pseudotuberculosis of the peritoneum has been met with. Dévé¹ has described multiple granulomas on the peritoneum enclosing fragments of hydatid membrane and hooklets in cases in which rupture of a hydatid cyst has occurred. The importance of this observation is to engender caution in making a naked-eye diagnosis of tuberculous peritonitis complicating an abdominal hydatid cyst. A case of pseudotuberculosis resembling tuberculous peritonitis, but due to the ova of a tape-worm, has been recorded by Helbing.

2. Loculated or encysted form of peritonitis. The loculated condition may be (a) suppurative (chronic tuberculosis, Osler; ulcerous, Allchin), or (b) ascitic. (a) In the most characteristic and serious form the intestines are matted together by adhesions and enclose collections of sero-fibrinous, turbid, or purulent exudation. In this form the tubercles increase in size, become confluent masses of caseous material surrounded by fibrin and adhesions, and by softening give rise to suppurating foci among the adherent coils of intestine. There may thus be numbers of intraperitoneal abscesses, the pus of which may erode the walls of the intestine and eventually lead to perforation, and as a result a fæcal fistula may form at the umbilicus. Purulent foci may also open into the vagina; in such cases the tubes were probably the original focus of infection. On attempting to unravel the intestines in such a case the bowel is easily torn, and it is sometimes difficult to be certain whether perforation was present during life or was made at the necropsy. The adhesions unite all the viscera; the omentum is thickened, contains caseous tubercles, and is usually rolled up into a firm cord below the transverse colon. The mesenteric glands are enlarged and caseous; they may soften and give rise to a circumscribed abscess, which in a case seen by me imitated a pancreatic cyst.

(b) In the ascitic form of loculated tuberculous peritonitis there is a transition between the ascitic and obliterative forms. A special though rare form of loculated tuberculous peritonitis is that in which a large cyst forms in the pelvis and so closely imitates an intra-abdominal cyst as to have been described as an "allantoic" cyst. More commonly encysted ascitic effusions of such a size as to be mistaken clinically for ovarian cystadenomas result from tuberculous peritonitis.

3. The obliterative (chronic fibroid tuberculosis, fibrous, or adhesive) form of tuberculous peritonitis may follow cure of the ascitic form or develop subacutely. The inflammatory process leads to universal adhesions which unite all the abdominal viscera inextricably to each other and to the abdominal wall; as a result there is no exudation. The tubercles tend to undergo the same fibrotic changes and show pigmentation. The matted intestines, the rolled up omentum, and masses of caseous glands embedded in adhesions give rise to the tumor-like masses felt through the abdominal wall. In exceptional instances large pendulous masses of caseous material in the peritoneal cavity, resembling "Perlsucht" in animals, have been described in man (Bizzozero,² MacCallum³).

¹ *Arch. de méd. expér. et d'anat. path.*, Paris, 1907, xix, 347.

² *Morgagni*, 1867, ix.

³ *Johns Hopkins Hospital Bulletin*, 1901, xii, 293.

Tuberculous peritonitis may, as already pointed out, be associated with cirrhosis of the liver; in the vast majority of cases the hepatic change is undoubtedly the older, but it is possible that some cases of interstitial change due to tuberculous infection of the liver have been set up by the existence of tuberculous peritonitis.

In some cases the serous membranes—pleura and peritoneum—seem especially liable to tuberculous infection, and may be found notably affected, although there is little evidence of tuberculosis elsewhere in the body. The lymphatic glands in the anterior mediastinum are infiltrated with tubercle as the result of infection conveyed to them from the abdomen.

Symptoms.—These vary very considerably according to the anatomical lesions present. In generalized miliary tuberculosis the infection of the peritoneum may be latent, and, there being no effusion, the peritoneal infection is not suspected during life unless there be abdominal pain or retention of urine and difficulty and pain in micturition. In local peritoneal tuberculosis there may be local pain and tenderness; this may be seen in the neighborhood of the vermiform appendix, and then naturally suggests appendicitis.

The anatomical forms into which diffuse tuberculous peritonitis has been divided may to some extent be correlated with corresponding groups of symptoms and physical signs.

1. In the ascitic form, in which miliary or discrete tubercles are scattered over the peritoneum, the characteristic feature is the presence of the effusion. The onset is usually gradual and preceded by failing health, loss of appetite, strength, and weight, with some abdominal discomfort or pain. The abdomen becomes swollen and tumid, often mainly tympanitic at first, and in children the condition may resemble that of chronic intestinal catarrh. As the case progresses the distention is gradually seen to be not entirely tympanitic, and evidence of free fluid becomes obvious and more distinct. In some cases the onset of ascites is sudden; this may depend on rupture of a caseous mesenteric gland into the peritoneal cavity. As a rule, the amount of fluid is not extreme, but in adults with existing hepatic cirrhosis the ascitic distention may be indistinguishable from that due to uncomplicated cirrhosis. The distended abdomen shows some dilated subcutaneous veins, and is tender on pressure, especially over any tumor-like masses due to enlarged glands. There are signs of fluid inside the abdomen. As the result of pressure exerted on the inferior vena cava by a large ascitic effusion, there may be some œdema of the feet and even a trace of albumin in the urine. The ascitic fluid may be clear, turbid, or even blood-stained, and may coagulate when removed. As pointed out elsewhere, the contained cells are mainly lymphocytes; tubercle bacilli are difficult to find, but injection of the fluid into guinea-pigs shows that the condition is tuberculous. The opsonic index of the effusion is lower than that of the blood serum.

- 2 (a) The suppurative or ulcerative form of encysted tuberculous peritonitis is the most characteristic. The onset of abdominal symptoms is, as in the ascitic form, preceded by failing health and an irregular temperature. The abdomen becomes swollen and tumid, often first in the lower part, and there is a complaint of abdominal discomfort and tenderness, and of gripping pains. The abdominal pain is made worse by walking and by jarring. The abdomen gradually becomes more distended, and the existence of fluid

is suspected, but being more or less encysted is often difficult to determine. On palpation the abdomen feels doughy and tumor-like masses due to enlarged glands, adherent coils of intestine, or the rolled up omentum may be felt through the abdominal wall. The thickened omentum forms a characteristic hard cord running transversely across the upper part of the abdomen; there is usually resonance above it, which enables it to be distinguished from the margin of the liver. The tumors are usually fixed, tender on pressure, and usually dull on percussion. The amount of distention and the distinctness with which these pseudo-tumors can be felt vary from time to time. This depends on changes in the amount of exudation and in the quantity of gas in the intestines. In advanced cases the umbilicus may become indurated and inflamed from an abscess pointing there; although this should always suggest the possibility of tuberculous peritonitis, it is not pathognomonic, for a localized abscess from pneumococcal infection may discharge at the umbilicus, and so may an abscess in puerperal infection of the peritoneum. Percussion gives rise to pain and shows irregular areas of resonance, due to tympanitic distention of intestinal coils, and of dullness due to localized exudation. Rectal or vaginal examination may reveal the presence of tumors due to glands, matted coils of intestine, or salpingitis.

(b) The ascitic form of encysted peritonitis shows a kind of transition from the ascitic form just described; the collection of serous fluid may simulate an abdominal cyst, especially in women, and may be confused with an ovarian cystadenoma; when confined to the pelvis and lower part of the abdomen it may imitate a urachal cyst, and, as pointed out elsewhere, cases of this kind have been described as "allantoic" cysts.

3. In the obliterative form the clinical manifestations are often vague. They may be mainly those due to interference with the functional activity of the alimentary canal, dyspepsia, flatulence, constipation, and pain; or there may be palpable tumors. Some cases with universal adhesions may not present any symptoms, but in others the intestinal movements may be so interfered with as to give rise to chronic obstruction. Symptoms of chronic intestinal obstruction may be brought about by the local cicatrization of peritoneal adhesions at one point; or in rare instances numerous peritoneal adhesions, no one of which produces definite obstruction, may so obstruct the intestines as to give rise to chronic obstruction; in such a case in a child the visible contracting coils of intestine may be seen through the abdominal wall. We will now pass to a more general consideration of the manifestations that may occur in the anatomical forms of tuberculous peritonitis.

The spleen and less often the liver may be enlarged. The writer has seen very considerable hepatic enlargement from fatty changes, and it may also be due to concomitant backward pressure. In boys with a patent funicular process between the peritoneal cavity and the tunica vaginalis testis tuberculous thickening may spread down the cord, and hydrocele may result.

The abdominal walls may show prominent subcutaneous veins, and the skin may become harsh, dry, and lose its elasticity. But with abdominal distention it may become shiny; in some instances it is pigmented, and sometimes pigmentation is also seen on the face, and may be regarded as indicating some degree of suprarenal insufficiency. There is often a drawn expression, and the face shows signs of grave illness. The condition of the

tongue varies; it may be red, irritable, and denuded of epithelium, or furred. Nausea, vomiting, and hiccough are rare. The motions may be colorless and fatty, especially in children, but this change occurs in other cachectic conditions, and, although suggestive, is certainly not pathognomonic of tuberculous peritonitis. From concomitant tuberculous ulceration there may be offensive diarrhœa and some blood in the stools. Bacteriological examination will show tubercle bacilli in the fæces. The temperature is raised in acute and suppurative cases, and may touch 104° F. In many cases there is but slight fever, and in the chronic forms the temperature may be subnormal. From abdominal distention and upward displacement of the diaphragm respiration is embarrassed, and the respiratory movements are rapid, shallow, and mainly costal. The pulse becomes rapid in acute cases, but in the chronic forms is not affected.

The urine often shows Ehrlich's diazo reaction; it does not contain the large quantities of indican seen in acute peritonitis. The urine is high-colored, but only exceptionally contains albumin. Micturition is frequently painful. In females tubercle bacilli may be found in the vaginal discharge.

The blood shows a moderate secondary anæmia, but from concentration of the blood due to ascites or purging there may be an apparent polycythemia. Leukocytosis is not part of the blood picture of the disease, but it occurs in a certain number of the cases (40 per cent, Emerson; 30 per cent., Shattuck; and 23 per cent., Cabot), and is the result of some complication or secondary infection. A high leukocyte count is more likely to be seen in children; thus the average count in 23 children was 16,435 (Rotch¹).

Jaundice from compression of the ducts by adhesions is an extremely rare event; Dujon² has reported an example. Jaundice may also be due to the pressure exerted by enlarged tuberculous glands in the portal fissure, but even this is a rare occurrence. Ulceration of a tuberculous gland into the hepatic artery has been known to produce an aneurism (Birkhardt and Schümann³).

Complications.—The complications of tuberculous peritonitis may be divided into those with abdominal symptoms and those due to more widespread tuberculosis. The abdominal complications may be mentioned first. Extensive tuberculous ulceration of the intestine, especially of the colon, may be suspected when there is diarrhœa with offensive stools and blood. Acute peritonitis may supervene as the result of rupture of a suppurating gland or from perforation of a tuberculous intestinal ulcer which, although very rare, does occur. The disease may remain latent until, from the onset of some complication, an acute abdominal crisis arises which may imitate acute peritonitis or acute intestinal obstruction. This acute onset may in some instances depend on rupture of a caseous gland in the mesentery or elsewhere into the peritoneal cavity; acute obstruction may be due to mechanical strangulation by a band, or, again, has been thought to be brought about by thrombosis in the tributaries of the mesenteric vessels and paralysis of the corresponding segment of intestine (Johnson⁴).

The thoracic complications are pulmonary tuberculosis and pleurisy, which may be bilateral. A tuberculous pleurisy may exist at the same

¹ *Journal of the American Medical Association*, 1903, xl, 69.

² *Procès-verbaux. XIX Congrès de chir.*, 1906, 166.

³ *Deut. Arch. f. klin. Med.*, Leipsic, 1907, xc, 288.

⁴ *Practitioner*, 1906, lxxvi, 332.

time as an active tuberculous peritonitis, precede it, or come on when the peritoneal lesion has ceased to be clinically manifest. In a patient with tuberculous peritonitis symptoms suggesting subacute intestinal obstruction, namely, constipation and vomiting, may be in reality due to the onset of tuberculous meningitis.

Diagnosis.—The presence of tuberculosis in the lungs, in the Fallopian tubes, or elsewhere in the body, is an important factor in the diagnosis. In children tuberculous infection of the peritoneum is much the commonest cause of ascites. In women the association of disease of the uterine adnexa should always suggest a tuberculous origin for ascites or chronic peritonitis.

In some rather rare cases there may for a time be a very considerable resemblance to typhoid fever. The most certain means of distinguishing between these two conditions is by a blood culture or by the agglutination reaction. When tuberculous peritonitis comes on acutely there may be very great difficulty in deciding whether the condition is due to pneumococcal or tuberculous infection.

It may be very difficult to avoid mistakes in the diagnosis between the ascites of tuberculous peritonitis and that of hepatic cirrhosis. In children hepatic cirrhosis is rare, and when it gives rise to ascites tuberculous peritonitis is very likely to be diagnosed. The writer¹ has published the notes of three children of one family, all of whom had portal cirrhosis and ascites and were operated upon with a fatal result; in two of them the operation was undertaken because tuberculous peritonitis had been diagnosed. In adults the diagnosis is not so difficult, as help is obtained from a history pointing to alcoholism or from the presence of tuberculosis in the lungs or elsewhere. But alcoholism disposes both to cirrhosis and to tuberculosis, and pulmonary tuberculosis is not uncommon in cirrhosis. Further, tuberculous peritonitis may supervene in the course of hepatic cirrhosis, and when this occurs the true nature of the ascites is not likely to be suspected until the fluid is withdrawn and examined; in tuberculous peritonitis the preponderating cells in the effusion are lymphocytes; in the ascites of cirrhosis, endothelial cells.

In simple chronic peritonitis there is an absence of fever, there is less pain, and examination of the fluid after tapping should enable a definite diagnosis to be made. Many cases formerly regarded as idiopathic ascites or simple chronic peritonitis were in reality tuberculous. Some assistance may be obtained by estimation of the opsonic index before and after abdominal massage, a considerable rise in the index pointing to tuberculosis.

Difficulty sometimes arises, especially in children, in deciding whether abdominal distention with fluid and the presence of tumors—omental and glandular—are due to tuberculosis or to malignant disease. In some cases a diagnosis is only made by opening the abdomen. As a rule, an irregular temperature and a history pointing to tuberculosis are in favor of tuberculous infection, which in children is of course much commoner than intra-abdominal sarcoma. In adults the presence of multiple tumors is more probably evidence of malignant disease. Cases of chronic intussusception in children have been ordinarily regarded as tuberculous peritonitis.

Prognosis.—This may be considered from several points of view: (a) as to immediate recovery; (b) as to relapses of the peritoneal infection;

¹ *Diseases of the Liver, Gall-bladder, and Bile Ducts*, 1095, p. 181.

(c) as to remote mechanical effects of adhesions, such as pain or intestinal obstruction.

(a) As to immediate recovery. The prognosis is different in the various forms of peritoneal tuberculosis. The disease may become arrested and the symptoms disappear without any manifest cause. The improvement may be permanent or only temporary. There can be no question that spontaneous cure occurs in a certain number of cases. For in cases in which successive laparotomies have been performed, the first for tuberculous peritonitis, the second for some other cause, all tubercles and adhesions have been proved to have disappeared. The prognosis is best in the discrete miliary form with ascites when uncomplicated, and worst in the form with numerous localized foci of purulent exudation. According to F. Taylor, tuberculous peritonitis is probably fatal in more than half the cases, but the general impression is that the outlook is more hopeful than this and that spontaneous cure occurs in 50 per cent. In estimating the outlook in an individual case, the condition of the general nutrition, the evidence of tuberculosis elsewhere, as in the lungs, the presence of diarrhoea, and of a raised temperature must be taken into account. An increased excretion of chlorides in the urine is said to be a sign of improvement. Wasting, diarrhoea, and persistent fever are bad signs. The prognosis is bad in the presence of complications such as extensive ulceration of the intestines, well-established pulmonary tuberculosis, generalized tuberculosis, meningitis, and evidence suggesting perforation of a suppurating mesenteric gland into the peritoneal cavity. It is said that the presence of pleurisy does not make the prognosis worse. An abdominal abscess is a grave sign, but recovery may follow discharge of pus from the umbilicus; a fecal fistula is nearly always fatal.

(b) A recurrence of abdominal tuberculosis occurs in a certain number of cases. This probably depends on the presence of some source of reinfection in the abdominal cavity—namely, in the intestine, lymphatic glands, vermiform appendix, or the Fallopian tubes. The importance of removing the local focus whenever possible has been shown by W. Mayo; thus in 26 cases in which the Fallopian tubes were removed, 25 recovered; 7 of these had previously undergone simple laparotomy for the cure of tuberculous peritonitis from one to four times, and in none was a further operation necessary after removal of the tubes.

(c) The remote mechanical effects of tuberculous peritonitis are due to the persistence of peritoneal adhesions which may give rise to internal strangulation of the intestine and acute obstruction. Undoubtedly a considerable proportion of the cases of strangulation by bands occur in patients who have suffered from tuberculous peritonitis, but the number of cases of tuberculous peritonitis in which acute obstruction is due to a band must be small. Since it has been shown that tubercles and peritoneal adhesions may completely disappear, the persistence of adhesions probably depends on a particularly severe form of peritonitis, or, more probably, on the persistence of a tuberculous focus in the peritoneal cavity.

Treatment.—This may be divided into (1) medicinal, (2) by injection of vaccines, (3) hygienic measures, and (4) by surgical means.

Medicinal.—The patient should be kept in bed in a well-lighted and airy room. The bowels should be regulated, any tendency to diarrhoea controlled, and constipation obviated. Offensive stools should be treated by guaiacol, which is less irritating than creosote, or if offensive and loose,

by salicylate of bismuth. Cod-liver oil and the syrup of the iodide of iron may be given with advantage, but the dose of cod-liver oil should be small at first and the stools should be examined to see that it is being assimilated. Iodine in solution has also been injected under the skin, and good results have been recorded. Mercurial ointment as a local application to the abdomen has been much used, and in the past was highly thought of; iodide in the form of liniment or tincture has been employed with the same object. Whether these applications really do good is doubtful, and it is at least possible that the good results seen after their use are due to improved hygiene, which permits spontaneous cure to take its natural course.

Injection of Vaccines.—Tuberculous peritonitis, being a local focus of tuberculosis, has been successfully treated by A. E. Wright's¹ method of therapeutic vaccines. Tubercle vaccine (Koch's new tuberculin) in doses of $\frac{1}{1000}$ mg. of the powdered vaccine is given, and is not increased above $\frac{1}{600}$ mg. For children, Riviere² advises much smaller doses, $\frac{1}{12000}$ to $\frac{1}{8000}$ mg. for a child of one year, $\frac{1}{4000}$ mg. for a child of five years, and $\frac{1}{3000}$ mg. for children of ten to twelve years. Latham has obtained good results from tuberculin in horse serum administered per rectum. Raw regards peritoneal tuberculosis as due to infection with the bovine form of *Bacillus tuberculosis*, and believes that the human and bovine forms are antagonistic. He therefore has good logical grounds for treating tuberculous peritonitis with tuberculin prepared from human bacilli. Further information on this question is desirable.

Hygienic Treatment.—Very satisfactory results follow moving the patient in the early stages into the country and especially to the seaside, with the adoption of open-air treatment. This course should be adopted as early as possible, and should be persisted in until recovery has been fully established.

Surgical Treatment.—The operative treatment really dates from 1862, when, as the result of what may perhaps be regarded as a fortunate error of diagnosis, the late Sir Spencer Wells opened the abdomen of a woman twenty-two years of age, but instead of an ovarian cystadenoma, as had been anticipated, found widespread peritoneal tuberculosis; the abdomen was emptied, and after a sharp attack of peritonitis the patient got well and remained so.

Treatment by laparotomy with simple evacuation of the ascitic fluid was thus started, and as long ago as 1890 König published 131 cases, with 84 recoveries. It was supposed that operation reduced the rather feeble vitality of the peritoneal tubercles, and thus led to their death. The way in which the withdrawal of fluid acts has been recently explained in the following manner: peritoneal tuberculosis being a local infection, the opsonic index of the ascitic fluid is lower than that of the blood plasma; hence, if the ascitic fluid be removed, it enables a fresh effusion from the blood to occur; the opsonic index of this recurrent effusion is higher than that of the original one, and therefore exerts a curative effect on the local tuberculous process (White³). If this were the only object to be aimed at, simple paracentesis should be equally effective, and probably cases of ascites really due to tuberculosis, although not recognized as such, have in the

¹ *Medical-Chirurgical Transactions*, London, 1906, lxxxix, 19.

² *British Medical Journal*, 1907, ii, 1131.

³ *Transactions of the Royal Academy of Medicine of Ireland*, 1906, xxiv, 410.

past been cured by simple paracentesis. Schulze has obtained good results by injecting oxygen through the cannula after tapping. The utility of laparotomy is, however, by no means confined to removal of the ascites; it is important to take away, when possible, the primary focus of tuberculous infection, such as the Fallopian tubes or the vermiform appendix, so as to prevent continued infection and a relapse.

The question whether laparotomy gives better results than medical treatment has been extensively discussed, and numerous statistics have been brought forward by surgeons and physicians. Elestratov collected 136 cases of tuberculous peritonitis treated medically, with 31.6 per cent. of recoveries, and 240 cases treated surgically, with 78.3 per cent. of recoveries. F. C. Shattuck¹ found that in 52 cases treated surgically the ultimate mortality was 37.5 per cent., and that in 46 cases treated medically the mortality was 68 per cent., but explains this by the frequency with which the cases in the latter series were complicated by tuberculosis elsewhere in the body. In Roersch's² collection of 358 operation cases, 253, or 71 per cent., recovered. On the other hand, Borchgrevink³ and Sutherland⁴ have published smaller parallel series of cases treated medically and surgically, showing a higher percentage of recoveries among the cases treated medically; thus their combined figures show that out of 44 cases treated medically, 36, or 82 per cent., recovered and out of 36 cases treated by laparotomy, 21, or 58 per cent., recovered. Results equal to those obtained by laparotomy have been claimed for simple paracentesis, or for paracentesis followed by injection of an emulsion of iodoform in glycerin (Schömann⁵). After König's results were published treatment by laparotomy became popular. Borchgrevink, Fenger, and others protested against the assumption that the good effects of laparotomy were necessarily due to the operation, and insisted that the natural course of the disease is to undergo spontaneous cure. Borchgrevink and Saltykow⁶ performed experiments on animals to show that laparotomy in artificially produced tuberculous peritonitis does not have any notable influence on the tubercles. At the present time opinion is still somewhat divided between these two views, but on the whole the pendulum has swung in the direction of the value of laparotomy under proper restrictions.

Patients to be submitted to laparotomy should be selected; those with effusion should be chosen, the dry cases and the ulcerative cases should be avoided, and extensive tuberculosis elsewhere should be regarded as a contra-indication to operation. The cases suitable for laparotomy are therefore those in which the prognosis of a spontaneous cure is favorable, and the question has naturally been much debated whether laparotomy really exerts a beneficial influence. It is probable that when employed in suitable cases laparotomy does accelerate a cure. Medical and hygienic treatment should be tried first, and if improvement follow, should be persisted in. If improvement does not occur, operation is indicated, unless there be some reason to the contrary. Shattuck advises that after a month

¹ *American Journal of the Medical Sciences*, Philadelphia, 1902, cxxiv, i.

² *Rev. de Chir.*, Paris, 1893, xiii, 531.

³ *Grenzgeb. d. Med. u. Chir.*, 1900, vi, 434.

⁴ *Clinical Journal*, London, 1902-3, xxi, 189.

⁵ *Zentralbl. f. Chir.*, Leipsic, 1904, xxxi, 1409, Orig.

⁶ *Arch. de méd. expér. et d'anat. path.*, Paris, 1903, xv, 571.

or six weeks without any evidence of improvement operative measures should be adopted, and that any sign of increase in the activity of the disease should lead to earlier operation.

TUMORS OF THE PERITONEUM.

The peritoneum, being much more extensive than the other serous membranes, and covering various organs especially liable to primary new-growth, is therefore more frequently the site of tumors, both primary and secondary, than the pleura and pericardium. The subject of new-growths of the peritoneum will be considered under the following heads:

1. Non-malignant tumors. (a) Solid. (b) Cystic.
2. Malignant growths. (a) Primary. (b) Secondary.

Non-malignant Tumors.

These are not so rare as primary malignant tumors of the peritoneum, but this is mainly due to the inclusion, among innocent tumors, of cysts which are commoner than the solid innocent growths.

Solid Non-malignant Tumors.—Fibromas.—In the first place circumscribed thickenings of the peritoneum due to attrition, such as the lamellar fibromas commonly seen on the surface of an enlarged spleen, or those due to chronic inflammation, such as the fibrous plaques sometimes present in chronic peritonitis, fibrosed remains of tubercles, and the small nodules in pseudotuberculosis of the peritoneum, must be excluded from consideration. A lamellar fibroma has been known to form around a piece of steel (Shattuck¹) that had worked its way out of the bowel, but a new formation due to such a definite cause is on rather a different footing from ordinary tumors. Fibrous tissue is also found in combination with fatty and mucous tissue in fibrolipomas and fibromyxomas.

Although not intraperitoneal tumors, attention may be directed to the interesting group of fibrous desmoid tumors of the anterior abdominal wall, which although innocent are often described as fibrosarcomas. They occur mainly in women, especially in multiparæ and during pregnancy, and it has been said that the protuberance of the abdomen favors their development. They are more often below than above the umbilicus; they are equally common on the right and left sides. They arise from the sheath of the rectus muscle, from the neighboring aponeuroses, from the iliac crest (Nélaton), from tendons (Doran), or possibly from the round ligament of the uterus. Like other innocent tumors, they may be multiple. Under the impression that they are fibrosarcomas, these tumors have been removed during pregnancy; this is not only unnecessary, but undesirable, as the abdominal wall is thus weakened. In 100 cases of connective-tissue tumors of the abdominal wall collected by Stavely,² 79 were in women; it is not surprising that these tumors have been mainly studied by gynecologists.

Pure fibromas are rare, they are usually single and of considerable size,

¹ *Transactions of the Pathological Society of London*, 1893, xliv, 151.

² *Philadelphia Medical Journal*, 1900, v, 638.

and are found in connection with the mesentery, omentum, or the retroperitoneal space. They probably arise from proliferation of preëxisting fibrous tissue in these positions, but Belkowsky¹ described a hard fibroma which he believed was derived from an appendix epiploica. A special group of fibromas arising from the subperitoneal tissues of the true pelvis has been described by Whitney.² The tumors are composed of oedematous fibrous tissue enclosing some endothelial cells, and may produce hernial protrusions in the perineum, ischiorectal region, labium, or scrotum. Of Whitney's 19 cases, 15 were females and 4 males. They may reach a very considerable size.

Fibromyomas, Myomas.—Some apparently fibrous tumors growing from the retroperitoneal space and from the root of the mesentery contain smooth muscular fibers, which may be derived from Treitz's muscle in the root of the mesentery, and are really fibromyomas. The tumor may pass between the layers of the mesentery and form a solid mesenteric tumor. In 13 cases of fibromyoma or myoma of the mesentery, collected by Doran,³ 10 were in women and 3 in men, thus disproving the view that these tumors are necessarily of uterine or broad ligament origin. Some of the fibromyomas described as growing from the pelvis probably start from the smooth muscular tissue in the broad ligament, and, though not actually growing from the uterus, closely resemble uterine fibromyomas and like them reach a very considerable size. It is conceivable that a subperitoneal uterine fibromyoma with a much attenuated pedicle might become detached and contract fresh adhesions elsewhere in the abdominal cavity. Fibromas and fibromyomas are encapsulated tumors and shell out readily; they may show mucoid degeneration with the formation of spaces in their substance, and so resemble cystic fibromyomas of the uterus. Fibromyomas, or possibly in rare instances myomas, of retroperitoneal origin may be multiple.

The *clinical aspects* of fibromas and fibromyomas depend upon the position of the tumor; those in the lower part of the abdomen will, in women, resemble uterine myomas. The special feature of the fibromas arising in the true pelvis—viz., the tendency to give rise to hernial protrusions—has already been referred to. A fibroma in the small omentum, as in Jackson Clarke's⁴ case, in which it had existed for four years, would be very difficult to diagnose.

In fibromas and fibromyomas of the mesentery the symptoms come on slowly, pain is absent in most instances, and intestinal obstruction, which is not uncommonly brought about by mesenteric cysts, does not seem to occur. Lockwood laid stress on the occurrence of irregular and inconstant areas of dulness and resonance over mesenteric tumors, but this sign is not always present (Doran). The correct diagnosis is very difficult, and these solid, innocent mesenteric tumors are very likely to be regarded as ovarian cysts or uterine fibromyomas.

The *treatment* is surgical, and consists in removal. Doran successfully removed a fibromyoma of the mesentery weighing 30 pounds; of 23 fibromas or fibromyomas of the mesentery removed by operation, 17 were cured (Bowers⁵).

¹ *Rev. méd. de la Suisse Romande*, Geneva, 1893, 431.

² *Annals of Surgery*, 1905, xli, 823.

³ *British Medical Journal*, 1904, ii, 1075.

⁴ *Transactions of the Pathological Society of London*, 1892, xliii, 60.

⁵ *Annals of Surgery*, 1906, xliv, 892.

Lipomas.—Lipomas may arise in the abdominal cavity (1) in connection with hernial orifices, the inguinal, femoral, or obturator rings, and in weak spots or defects in the linea alba. Overgrowth of fat in these positions may exert traction, and so pull out a process of the parietal peritoneum and thus give rise to hernia. The inguinal form of the hernial lipomas, which may be double, may reach a large size, whilst the mesial lipomas in the linea alba, which, like the other varieties, may imitate a true hernia, are the commonest. They may give rise to troublesome symptoms, such as pain and vomiting, which, unless the cause is detected, are difficult to explain (Anderson¹); they will not be referred to further here. (2) In rare instances they arise from giant-growth of an appendix epiploica; Gruber² recorded a large pedunculated lipoma which formed the only content of a left scrotal hernia. (3) From the fat around the kidneys and in the iliac fossæ; they lie under the prerenal layer of fascia which separates them from the colon, intestines, and pancreas. These lipomas are rather more common on the right side; and may remain retroperitoneal, pushing the abdominal contents as a whole in front of them, or may work their way between the layers of the mesentery, mesocolon, omentum, or, in very rare instances, of the broad ligament (Middelschulte,³ Peyrot,⁴ Treves,⁵ Borrmann⁶), and so become more or less pedunculated. The transverse colon may become surrounded by the adipose growth. They are usually single, but may show signs of fusion of originally separate tumors. They may be composed of fat only or of fat mixed with fibrous tissue, show myxomatous degeneration, calcareous infiltration, or very rarely contain cartilage. In very exceptional instances sarcomatous change has supervened. They may be encapsuled like the ordinary fatty tumor of the subcutaneous fat, or in other instances, like diffuse lipoma, shade off into the retroperitoneal fat from which they arise. They are often of great size, most of the recorded instances have been found to weigh over twenty pounds, and about 6 have been more than fifty pounds. In Buckner's⁷ extraordinary case, a fibrolipoma arising in the subperitoneal tissue of the true pelvis was estimated to weigh 268 pounds.

Etiology.—The large lipomas, which are decidedly rare, are more frequent in women than in men; thus, in 41 cases tabulated by Adami,⁸ 25 were in women and 16 in men. The usual age is between thirty and fifty years, but isolated examples have been recorded in children. Lauwers described a case in a child aged seven years; the child was emaciated and weighed twenty pounds only, while the tumor, which had been detected when the child was two weeks old, weighed six pounds. They are not necessarily associated with general obesity. Proust and Treves⁹ have collected 89 cases, of which 14 were parietal, 38 perirenal and 37 mesenteric. Ten were multiple and the remainder single tumors. Of 84 cases, 61 or 73 per cent. were males and 23 or 27 per cent. females.

Physical Signs.—Unless of a large size, they are not likely to be detected, as from their soft consistency they are difficult to feel. They grow slowly,

¹ *British Medical Journal*, 1896, ii, 1087.

² *Virchow's Archiv*, 1885, cii, 541.

³ *Inaug. Diss.*, Greifswald, 1884.

⁴ *Bulletin of the Society of Anatomy*, Paris, 1875, 170.

⁵ *Transactions of the Clinical Society of London*, 1893, xxvi, 101.

⁶ *Virchow's Archiv*, 1907, clxxxix, 436.

⁷ Quoted by Whitney, *Annals of Surgery*, 1905, xli, 823.

⁸ *Montreal Medical Journal*, 1897, xxv, 529, 620.

⁹ *Rev. gyn. et de chir. abdom.*, 1908, xii, 93.

so that the increase in size of the abdomen is gradual. When palpable, the tumor is soft, smooth, free from tenderness, and semifluctuating, so that ascites, either general or encysted, is commonly suspected, although ovarian or hydatid cysts have been diagnosed. As growth proceeds, the abdominal distention, which is mainly in the anteroposterior diameter, increases, the diaphragm is pushed up and the contents of the thorax pressed upon. The later stages are attended with general wasting and œdema of the feet; malignant disease may then appear probable.

Symptoms.—The progress of the disease is slow, the general health and nutrition are well maintained, and symptoms do not appear for a long time. They include a feeling of weight, fulness, and discomfort from abdominal distention, sometimes gastro-intestinal symptoms from pressure on the stomach and intestines, inconvenience from pressure on the urinary bladder, and sooner or later dyspnoea from the upward displacement of the diaphragm.

Diagnosis.—The distinction from ascites is extremely difficult, and the existence of such a rare condition as a lipoma is only likely to be suspected after the abdomen has been unsuccessfully tapped. From malignant disease the slow course of the disease is the main distinction. From ovarian and hydatid cysts they differ in their softness and indefinite outlines. Difficulty has occurred in distinguishing them from large lipomas of the anterior abdominal wall.

Prognosis.—This depends upon their being operated upon before they have reached a very large size, for if left alone the patients gradually become emaciated and die. In 26 cases in which the tumor was wholly or partially removed, recovery followed in 12, with recurrence in one of these (Adami). Recurrence after apparently complete removal has been recorded by Johnstone.¹

Treatment.—The only radical method of dealing with these tumors is surgical, but the results of removal differ in those that are entirely retroperitoneal and in those that are pedunculated or situated in the omentum. Removal of a retroperitoneal lipoma has, in some cases, deprived a considerable extent of the intestine, which is carried in front of the tumor, of its blood supply, and so led to gangrene or necessitated resection of part of the intestine. In 31 retroperitoneal lipomas operated on the mortality was 51.6 per cent. (Reynolds and Wadsworth²). As an entirely retroperitoneal tumor has only been successfully removed in a very few instances (Doran), an exploratory operation should be undertaken, and if the lipoma is entirely or mainly retroperitoneal, the perirenal space should be opened external to the colon, so as to avoid interfering with the vessels running to the intestines.

Angioma.—Angioma has been met with in very rare instances, but it is probable that the line dividing them from angiosarcoma is rather thin. In Julliard's³ case a cavernous angioma of the mesentery was as large as an adult's head, weighed 4700 grams, and produced intestinal obstruction. A cavernous angioma of the ascending layer of the transverse mesocolon was diagnosed as a twisted mesenteric cyst (Wagener⁴). Lane⁵ successfully removed a large degenerating cavernous angioma from the peritoneum of a

¹ *British Medical Journal*, 1907, ii, 950. ² *Annals of Surgery*, 1906, xlv, 61.

³ *Rev. de gyn. et de chir. abdom.*, 1904, viii, 229.

⁴ *Zentralbl. f. Gynäk.*, Leipsic, 1904, xxviii, 1600.

⁵ *Transactions of the Clinical Society of London*, 1893, xxvi, 5.

child aged seven years. The relation of these rare and large angiomas to angiosarcomas is a point of considerable importance.

Myxoma.—Myxomatous degeneration, of course, occurs in fibromas, myomas, and lipomas, and in some instances the condition may be so advanced as to appear to be a pure myxoma.

Cysts.—*Parasitic.* Hydatids, cysticercus.

Non-parasitic. Mesenteric, omental, sanguineous, retroperitoneal hæmatoma, dermoid, retroperitoneal, urachal, conditions simulating cysts.

Hydatid Disease of the Peritoneum.—The cystic or bladder stage of the *Tænia echinococcus* (vide vol. i, p. 576), which exists as a tape-worm in the intestine of the dog, fox, wolf, and jackal, sometimes infects the peritoneum. Although very striking, this condition is not common; thus, in 1897 cases of hydatid disease, tabulated by Davies Thomas,¹ the peritoneum, omentum, and mesentery were affected primarily in 26, or 1.4 per cent., only. No statistics exist to show whether hydatid disease of the peritoneum was commoner in the early part of the last century, when Bright gave such a graphic account of its clinical features, but the practice then in vogue of tapping these cysts through the abdominal walls with a trocar offered much greater facilities for the escape of daughter cysts, brood capsules, and scolices into the peritoneal cavity and so to infection than the more radical, although safer, surgical measures now employed.

The method by which infection of the peritoneum with hydatid cysts takes place has given rise to some discussion, and it is probable that the method is not always the same. In cases with a large number of cysts implanted on the peritoneum the most probable explanation is that rupture or leakage from a single and primary cyst, usually in the liver, has infected the peritoneum. Thus tapping a cyst with a trocar may allow small, daughter cysts, scolices, and brood capsules to escape into the peritoneal cavity and so lead to multiple infection of the peritoneum. It appears to take about two years for cysts thus implanted on the peritoneum to become sufficiently large to give rise to signs and symptoms. Dévé² has shown that the presence of bile mixed with ascitic fluid does not prevent the secondary infection of the peritoneum with hydatid cysts. On the other hand, in instances in which a single cyst or only a few cysts occur in the retroperitoneal tissue, a very common position being between the prostate gland and the rectovesical fascia, the embryo has probably burrowed its way from the bowel or from the bloodvessels in the neighborhood toward the peritoneum. Cranwell,³ who has collected 56 examples of retrovesical hydatid cysts in males, believes that most of the retrovesical cysts are due to peritoneal infection, a daughter cyst or scolex being able to undergo an apparent process of migration from the peritoneal cavity into the extraperitoneal tissues.

Morbid Anatomy.—The number of cysts may be enormous when the infection is secondary to rupture of a preëxisting cyst; the omentum, mesentery, and visceral peritoneum may be almost universally affected, but the omentum and pelvis are the most frequent sites. The cysts may be of almost any size, from a pinhead upward; when single or few, the cysts tend to be bigger

¹ *Hydatid Disease*, Adelaide, 1884.

² *Compt. rend. Soc. biol.*, Paris, 1903, iv, 75.

³ *Rev. de gyn. et de chir. abdom.*, Paris, 1907, xi, 606.

than when large numbers are present. The cysts may project from the surface of the viscera, be sessile on the peritoneum, especially on the omentum, mesentery, and pelvis, be pedunculated, and in rare instances a cyst may lie free in the abdominal cavity. Pedunculated hydatids may be sterile, probably from a deficient supply of nourishment. The cysts may be separate or crowded together. According to Stirling and Verco,¹ adhesions around uninfamed cysts occur only after injury or operative interference.

Over the surface of the cysts chronic peritonitis may be set up by mechanical irritation, and may lead to the formation of a thick fibrous capsule over dependent or other cysts, such as those in Douglas' pouch, exposed to friction; calcification may subsequently occur. A rare but interesting condition of "pseudotuberculosis" of the peritoneum may follow rupture of a cyst, and consists in small nodular granulomas, containing hooklets or pieces of hydatid membrane scattered over the surface of the peritoneum. These granulomas show large giant cells surrounding pieces of laminated membrane. The condition may readily be mistaken for tuberculous peritonitis.

Suppuration may take place in one or more of the cysts and set up localized abscess formation or general peritonitis. In a case reported by J. W. Ogle² a collection of suppurating cysts in the omentum set up pylephlebitis and multiple abscesses in the liver.

Clinical Aspect.—The enlargement of the abdomen is gradual in onset, and may continue for years; thus in one of Bright's³ cases it had lasted for at least ten years. There may either be a diffuse swelling, or separate cysts can be made out, not only by palpation, but with the eye. The cystic tumors are often so firm as to suggest solid growths, they are freely movable, alter their position with that of the body, and are not obviously connected with any of the abdominal viscera. A hydatid thrill is rare, and as it may be present over other cysts, such as ovarian or mesenteric, and even in ascites, too much stress must not be laid on it. When the abdomen becomes very greatly distended the diaphragm is pressed up and respiration is much embarrassed, whilst pressure on the stomach and intestines may lead to dyspepsia, constipation, or, in very rare instances, to intestinal obstruction (Hutchinson⁴). As the result of pressure on the inferior vena cava, the superficial abdominal veins may become very prominent and the urine scanty and high-colored. A retrovesical cyst in the pelvis may press on the rectum, bladder, and ureters, and may thus imitate a hypertrophied prostate or lead to hydronephrosis, consecutive kidney disease, and pyelonephritis. Out of 24 autopsies on such cases, the ureters were dilated in 11 (Cranwell). There is vesical pain, frequent micturition, constipation, and a feeling of weight in the perineum.

The multiplicity of the cysts renders these cases very difficult to deal with surgically, and they must be regarded as distinctly unfavorable.

Diagnosis.—If multiple and cystic tumors can be felt, the true solution is not difficult, but when the patient is a woman and one or two cysts only are palpable, the disease may be thought to be ovarian cystadenomas or even uterine fibromyomas. The history that a hydatid cyst of the liver

¹ *System of Medicine* (Allbutt and Rolleston), 1907, vol. ii, Part 2, p. 999.

² *St. George's Hospital Reports*, 1867, ii, 347.

³ *Clinical Memoirs on Abdominal Tumors*. New Sydenham Society. Edited by G. H. Barlow, 1860.

⁴ *Medico-Chirurgical Transactions*, London, 1894, lxxvii, 133.

has existed or has been tapped would be strongly in favor of hydatid infection of the peritoneum. From multiple abdominal malignant disease the slow course of the affection and evidence that the tumors are cystic, should distinguish it. Exploratory puncture should not be undertaken to clear up the diagnosis, as serious symptoms and even death may follow escape of hydatid fluid into the peritoneal cavity. If it be done, the discovery of scolices, hooklets, or of the characteristic laminated ectocyst will make it certain that a hydatid had been aspirated; chemical analysis, however, does not settle the question, for some hydronephroses contain fluid of a very similar composition (Stirling and Vereo). In some cases of hydatid infection there is eosinophilia, but this sign is so inconstant that its absence does not militate against hydatid disease.

Treatment.—When a single cyst is thought to exist, it should be fully exposed and dealt with surgically; when the cysts are manifestly multiple, operation is still the only available means of treatment, but the result is not likely to be so satisfactory as with a single cyst. In no case should a cyst be tapped through the abdominal wall. A single cyst in the pelvis may be opened through the perineum or vagina, but Cranwell recommends Tuffier's method of a median incision above the pubes. The outlook in these retrovesical hydatid cysts is grave, as out of 56 cases collected by Cranwell, 25 proved fatal.

Cysticercus Cellulosæ.—*Cysticercus cellulosæ* is, in rare instances, met with in great numbers inside the abdomen, chiefly in the mesentery, but this condition gives rise either to insignificant or to no clinical symptoms.

Mesenteric Cysts.—Various forms of cysts may be found in the mesentery. Some of them—hydatid, sanguineous, and “dermoid”—may occur in other parts of the abdomen, and are described elsewhere; attention will, therefore, be especially directed to those mesenteric cysts which are either not found at all elsewhere or only very rarely. These mesenteric cysts are nearly always close to the intestine, and from the nature of their contents have been described as lymphatic, serous, or chylous. The term “mesenteric cyst” has sometimes been applied to cysts in the omentum and mesocolon, but it is better to limit it to cysts in the mesentery of the small intestine and to employ it solely as descriptive of the position of the cyst.

Pathogeny.—Several views have been put forward as to the origin of these cysts. It has been thought that they are due to dilatation of lymphatics and lacteals, or even of lymphatic glands. Possibly some are of this nature, but some of those more recently examined have been found to contain smooth muscular fibers in their walls, and have been regarded as embryonic in origin (Dowd¹) and as sequestrations from the intestine or its neighborhood, some from mesoblastic tissue with fibrous or muscular walls or both; some from the hypoblast as well, and showing epithelial structures; the nature of the contents is largely a matter of chance. Thus chyle or blood might escape into a cyst with serous contents and so give rise to a so-called chylous or sanguineous cyst. The formation of these cysts would then be allied to obliteration of a Meckel's diverticulum at its junction with the ileum, while part of the distal portion remains pervious; Terrier and Lecène² have collected 18 cases of “enteroid” or juxta-intestinal cysts which they regard

¹ *Annals of Surgery*, 1900, xxxii, 515.

² *Rev. de chir.*, Paris, 1904, xxix, 161.

as derived from the remains of Meckel's diverticulum. It has been suggested that some mesenteric cysts are the outcome of other diverticula of the small intestine that have lost their continuity with the lumen of the alimentary canal, the snaring off of pieces of the wall of the intestine having occurred during foetal life. It has been shown experimentally by Carnot¹ that pieces of gastric mucous membrane of the stomach, urinary bladder, and gall-bladder, when implanted on the intestine, give rise to cystic formations. A case of a mesenteric cyst containing well-formed gastric mucous membrane with parietal cells has been observed (Walker-Hall²). On the other hand, cysts in the mesentery may have originated behind the peritoneum, and, having migrated between the layers of the mesentery, even come into contact with the intestine.

Pathological Anatomy.—These cysts are usually unilocular, but occasionally multilocular cysts have been met with. The cysts may project from the free border of the intestine or from its mesenteric border and may then pass between the layers of the mesentery. Cysts may be embedded in the layers of the mesentery without any direct connection with the intestine. Their surface is covered by peritoneum and their walls are composed of fibrous tissue, and may contain smooth muscular fibers very irregularly arranged, and some fat. There may be no layer of epithelium on the interior of the cysts which may be lined by granulation tissue. In some instances endothelium has been described, and in others columnar epithelium.

The contents may be clear and serous, containing albumin and cholesterin; turbid, with red or white blood corpuscles and albumin; viscid, with mucin; chylous, with the chemical composition of chyle. In very rare instances gas has been found in multiple cysts connected with the intestine; these formations have been regarded as homologous with the air bladders of fishes which secrete gas from the blood (Mair³).

Physical Signs.—The cyst is round, well-defined, tense, elastic, movable, is usually on the right side of the abdomen a little below the umbilicus, and is not connected with any of the viscera except the intestines. It is dull on percussion, is surrounded by resonant intestine, and there may be a band of resonance, due to the attached piece of intestine running across it.

Symptoms.—A sudden attack of abdominal pain is usually the first indication of the existence of a mesenteric cyst; there may be recurrent attacks of colic and vomiting; while in a number of the instances acute intestinal obstruction has resulted from kinking of the intestine. On the other hand the symptoms may be those of gastro-enteritis.

Diagnosis is often difficult, and when the cyst is detected, must be made from a distended gall-bladder, hydatid cysts hanging down from the liver, or in the peritoneum, floating kidney, ovarian or parovarian cysts of small size with a long pedicle, solid tumors of the mesentery or omentum, pancreatic cysts which are much larger, and even an appendicular abscess. No exploration tapping for diagnostic purposes is permissible.

Treatment is surgical, and consists in the enucleation of the cyst when this is possible, if not in drainage. The practice of tapping the cyst through the abdominal wall is dangerous, and should not be countenanced. These cysts should be operated upon without waiting for any urgent symptoms.

¹ *Arch. de méd. expér. et d'anat. path.*, Paris, 1905, xvii, 273.

² *Journal of Pathology and Bacteriology*, Cambridge, 1908, xii, 128.

³ *Ibid.*, 433.

Omental Cysts.—The omentum may be the seat of cysts of various kinds, such as hydatid and "dermoid," which are referred to elsewhere. In addition, cysts of rather doubtful origin have been met with. Cripps¹ and Berry² have recorded multiple serous cysts, possibly due to lymphatic obstruction. Hellier's³ case of lymphangioma of the great omentum appears to have been of the same nature. The large omental cysts may imitate localized tuberculous peritonitis, or in women, ovarian cysts with a long pedicle. Gairdner,⁴ Braithwaite,⁵ Mathews⁶ and others have described large single cysts with clear contents. Possibly these single cysts may be cystic lymphangiomas. Hemorrhage may take place into these omental cysts, as in Jacobi's⁷ case, but cysts of this kind must be distinguished from the sanguineous peritoneal cysts which are the result of hemorrhage. They have naturally been regarded as due to congenital aberrations, and possibly some are, like the retroperitoneal cysts, derived from the Wolffian body. Of 22 cases quoted by Fort,⁸ 50 per cent. were under ten years and 65 per cent. under twenty years of age, while three-fourths of this series were females.

The condition does not appear ever to have been definitely diagnosed before the abdomen was opened. Symptoms are not prominent in the early stages of growth, and pain usually leads to examination of the abdomen and the detection of a cyst which is superficial in position and usually movable to a limited extent.

Diagnosis.—Lipomas are the only solid tumors likely to be confused with omental cysts. Other cysts, such as hydatids, mesenteric, pancreatic, and, in women, ovarian cystadenomas with a long pedicle, localized tuberculous peritonitis, and even floating kidneys, aortic aneurism, and large cysts of the spleen (Powers⁹), have been mentioned as conditions which must be taken into consideration in the diagnosis.

Peritoneal Sanguineous Cysts.—Intra-abdominal cysts containing blood may be of very various kinds. Thus hemorrhage may take place into pre-existing cysts, for example, as the result of twisting of the pedicle of an ovarian cystadenoma, or into a mesenteric cyst. Hemorrhage may occur into a soft sarcoma, and malignant tumors of the suprarenals may contain more blood than growth. Collections of blood in the female pelvis, which are generally the result of an extra-uterine gestation, ruptured or false aneurisms, and angiomas, need only be mentioned in order to be distinguished from a fairly definite group of cases, the peritoneal sanguineous cysts (Fisher¹⁰). There is, however, very little doubt that many so-called pancreatic cysts with blood-stained contents are the same as the sanguineous cysts found elsewhere in the peritoneal cavity, and are hemorrhagic effusions into the lesser sac of the peritoneum and therefore peripancreatic, not pancreatic. Jordan Lloyd¹¹ described cases of traumatic hemorrhage into the lesser sac of the peritoneum.

¹ *Transactions of the Pathological Society of London*, 1897, xlviii, 85.

² *Ibid.*, 105.

³ *British Medical Journal*, 1904, ii, 1311.

⁴ *Transactions of the Pathological Society of London*, 1852, iii, 374.

⁵ *Lancet*, 1898, ii, 1472.

⁶ *British Medical Journal*, 1905, ii, 1642.

⁷ *Transactions of the Association of American Physicians*, 1901, xvi, 232.

⁸ *Annals of Surgery*, 1907, xlv, 382.

⁹ *Transactions of the American Surgical Association*, 1905, xxiii, 255.

¹⁰ *Guy's Hospital Reports*, 1893, xlix, 275.

¹¹ *British Medical Journal*, 1892, ii, 1051.

Morbid Anatomy.—Peritoneal sanguineous cysts may also be found in the great omentum, gastrohepatic omentum, mesentery, mesocolon, or arising from the peritoneum lining the back of the abdomen. The writer examined postmortem a case of sanguineous cyst arising in the pelvis in a man. The walls of the cysts are composed of well-formed, fibrous tissue and may contain some patches of blood pigment, but do not show any evidence of the laminated membrane characteristic of the ectocyst of a hydatid cyst, and the contents do not contain hooklets. The inside is usually lined by adherent shaggy fibrin.

The fluid in the cysts is reddish brown and contains blood corpuscles and in some instances cholesterol. In cysts in contact with the pancreas the fluid contains pancreatic ferments which have been able to pass through the damaged walls of that organ. Blood clot in various stages of change may be found in these cysts. Large nucleated cells—probably endothelial and derived from the peritoneum—also occur in the fluid.

Pathology.—The generally accepted view is that these cysts are the result of traumatic hæmatomas, but as the cyst may not become manifest until some time has elapsed since the injury, it has been suggested that local peritonitis first occurs and that from the newly formed vessels hemorrhage occurs—a pachyperitonitis hemorrhagica. The position of these cysts—in the omentum, mesentery, and other spaces into which hemorrhage may occur and then remain encysted—is in favor of the origin from a hæmatoma.

Clinical Features.—The cyst is much commoner in the upper than in the lower half of the abdomen, and is usually in the middle line. It forms a large, tense tumor, and may have pulsation conducted to it from the aorta. It can be shown by percussion to be distinct from the liver. Symptoms are less marked than in the case of the pancreatic or peripancreatic cysts, and after reaching a considerable size the tumor may remain unaltered for years.

Diagnosis of peritoneal sanguineous cysts other than those around the pancreas is very difficult. This is mainly due to their rarity. The existence of such a cyst might be suspected in the light of a recent injury, but this diagnosis could not be made with confidence.

Treatment is surgical and consists in draining the cyst. Excision is rarely possible, as the walls are so often part of the peritoneum forming the mesentery, lesser omentum, or mesocolon. Cure has followed tapping, but it is dangerous and should not be attempted until the cyst is exposed by laparotomy.

Retroperitoneal hæmatomas are rare; they may be briefly referred to here. They may be due to traumatism, such as rupture of the kidney, bursting of an abdominal aneurism, or leakage from a hemorrhagic retroperitoneal or renal sarcoma. In some instances the source of the blood is not clear. The blood tracks down behind the peritoneum toward the pelvis, and may form a distinct tumor accompanied by pain. On the right side such a tumor may suggest appendicitis. Mursell¹ has operated successfully on a case in which the symptoms were those of acute intestinal obstruction.

Dermoid Cysts.—Dermoid cysts in rare instances occur in the peritoneum without any evidence that they were directly derived from the ovaries. Thus,

¹ *British Medical Journal*, 1907, ii, 950.

single "dermoid cysts," or, more strictly, embryomas, for they are really teratomas, have been found between the layers of the mesentery, omentum, and transverse mesocolon, or in the retroperitoneal space. In some instances however, a "dermoid" cyst originating in the ovary may become detached and subsequently contract adhesions elsewhere. Dermoid cysts in the abdominal cavity are almost, if not entirely, restricted to the female sex.

Multiple "dermoid" cysts may be due to rupture of an ovarian embryoma with the resulting implantation of fragments over the peritoneum; the multiple growths are not malignant, but resemble the implantations sometimes associated with ovarian papillomas. Suppuration occasionally occurs in these cysts, and in rare instances, of which Montgomery¹ has collected 10 cases, the teratomas may become malignant.

Retroperitoneal Cysts.—Cysts manifestly arising in viscera behind the peritoneum, the kidneys, adrenals, and pancreas, are not included in this category. Although far from common, cysts of various kinds may be found in the subperitoneal connective tissue; for example, in rare instances, hydatid cysts. A multilocular cyst, structurally resembling an ovarian cystadenoma, may occur in the retroperitoneal tissues of men as well as of women. These cysts may arise close to the kidney and be mistaken for a floating kidney, a hydronephrosis, or on the left side for a primary neoplasm of the tail of the pancreas. Severe attacks of pain, simulating Dietl's crises, may occur; in Elder's² case the pain imitated that of appendicitis. The cysts may pass between the layers of the mesentery, omentum, mesocolon, or mesorectum, and in all probability are derived from sequestrations of the Wolffian body. They may contain several pints of fluid. The multilocular cysts may become malignant and give rise to metastases.

Among rare conditions cystic dilatation of the receptaculum chyli and collection of fluid in a subcæcal pouch, the mouth of which has been closed, may be mentioned.

Urachal Cysts.—The urachus, which runs in the middle line in front of the peritoneum from the apex of the urinary bladder to the umbilicus, may be partially pervious; thus minute cystic dilatations were found to exist in 24 out of 74 cases specially examined by Wutz.³ In rare cases cysts of considerable size and containing many pints of fluid may result from dilatation of part of the urachal canal. In some instances the cysts communicate with the bladder; infection may then spread into the cyst, and so account for a purulent condition of the contents; in other instances there is no communication between the cavities of the cyst and of the urinary bladder, the two being then connected by an impervious cord. The contents may be clear, turbid, blood-stained, or purulent. The cysts are usually in front of the peritoneum and adherent to the umbilicus and anterior abdominal wall, but may be intraperitoneal; the latter position is explained by a persistence of the mesentery of the urachus which is present in fetal life (Delore and Cotte⁴). The wall of the cyst contains bundles of smooth muscular fibers, elastic and fibrous tissue, and may show epithelium resembling that of the bladder, but this may be detached. The cysts are usually unilocular, but in Doran's⁵ case there were two cysts; they have been described by Tait, Doran, Delore

¹ *Journal of Experimental Medicine*, 1898, iii, 259.

² *Montreal Medical Journal*, 1905, xxxiv, 894.

³ *Virchow's Archiv*, 1883, xcii, 387.

⁴ *Rev. de chir.*, Paris, 1906, 403.

⁵ *Medico-Chirurgical Transactions*, London, 1898, lxxxi, 301.

and Cotte, Weiser,¹ and others. Weiser tabulated 89 cases, but included 19 cases recorded by Tait and Robinson, which were probably examples of encysted tuberculous peritonitis (Doran), so that the number of authentic cases on record is not so great. They occur chiefly in women, but have been met with in men and in children. A case in a man under G. R. Turner, in St. George's Hospital, was diagnosed correctly before operation. In Weiser's 89 cases females were about three times more often affected than males. The cysts are in the middle line between the umbilicus and the pubes; they may be soft and fluctuating, resembling a distended urinary bladder, or firm and appear to be solid. There may be pain in the lower part of the abdomen, probably from associated local peritonitis. They may imitate encysted peritonitis, especially of tuberculous origin, and in women may be mistaken for ovarian cystadenomas.

The *treatment* of urachal cysts is surgical, in some instances they can be dissected out, in others the only available course is drainage. It is important that the relation of the cyst to the bladder and the possibility of a communication between the two should be borne in mind, otherwise a troublesome urinary fistula may follow operation. Tapping the cysts is only a palliative measure, as the fluid is prone to accumulate again.

"Allantoic Cysts."—In 1887 Lawson Tait² described 12 cases showing a large cystic cavity occupying the whole of the pelvis and the middle and lower part of the abdomen; Byron Robinson³ in 1891 added 6 more cases. They suggested that the cysts were derived from the allantois; it appears from Doran's criticism, however, that the cases were really examples of encysted peritonitis, usually, if not always, tuberculous, and not an extreme form of urachal cyst.

Conditions Simulating Peritoneal Cysts.—Under this heading reference may be made in the briefest manner to conditions, very different in their nature, which may imitate peritoneal cysts. Thus clinically encysted peritonitis may simulate a true cyst, and pathologically small collections of serous fluid may become surrounded by fibrous adhesions and form single or multiple cysts. Kelly and Hurdon⁴ figure cysts of this nature adherent to the tip of the vermiform appendix.

By *pseudomyxoma* of the peritoneum is meant the condition of numerous encysted collections of gelatinous material in the peritoneum which follows rupture of an ovarian cystadenoma. This curious condition was regarded by Werth⁵ as the result of peritonitis following rupture of an ordinary ovarian cyst. Pseudomyxoma of the peritoneum has been recorded in a male, and has been thought to be due to the escape of the contents of a mucocoele of the vermiform appendix (Hueter,⁶ McConnell⁷). It may be confused with colloid cancer and with primary tumors derived from the Wolffian body and structurally resembling ovarian cystadenomas.

¹ *Annals of Surgery*, 1906, xliv, 529.

² *British Journal of Gynecology*, 1887, ii, 328.

³ *Annals of Surgery*, 1891, xiv, 350.

⁴ *The Vermiform Appendix and its Diseases*, 1905, 311.

⁵ *Arch. f. Gynäk.*, Berlin, 1884, xxiv, 103.

⁶ *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1907, xli, 3.

⁷ *International Clinics*, 1907, 17th series, vol. iv, p 153.

Malignant Tumors.

Malignant growths are more often met with in the peritoneal cavity than innocent tumors, but this depends on the frequency of secondary infection of the peritoneum, for whilst primary malignant disease and innocent neoplasms of the peritoneum are both rare, primary malignant disease is the more exceptional of the two.

Primary Malignant Diseases.—Primary malignant disease of the peritoneum may conceivably arise in several structures, such as (1) the endothelium of the serous membrane, of its lymphatics and bloodvessels; (2) the subserous connective tissues, especially around the spine and back of the abdomen, the periosteum of the spinal column and pelvis; and (3) the remains of the Wolffian bodies, Müllerian ducts, accessory adrenal bodies, or even from teratomas that have undergone malignant transformation.

The tumor growth may occupy the omentum or, more rarely, a peritoneal fold, such as the mesentery or the gastrohepatic omentum, and be thus to all intents and purposes intraperitoneal. In other cases the growth extends behind the peritoneum and to a slight extent displaces the abdominal contents forward; these are cases of retroperitoneal sarcoma. In some instances a growth at first definitely retroperitoneal passes forward, becomes pedunculated, and therefore partly intraperitoneal. Thus, although it might appear to be more logical to describe separately malignant disease (*a*) arising from the peritoneum itself, and (*b*) originating in the tissues (as apart from the organs) of the retroperitoneal space, any such distinction would in reality be artificial.

Morbid Anatomy.—Primary malignant tumors of the peritoneum and retroperitoneal space belong to the group sarcoma or endothelioma. Formerly primary carcinoma of the peritoneum was commonly described; alveolar growths resembling carcinoma histologically undoubtedly occur, but are now regarded as endotheliomas and arising in endothelium, either of a lymphatic or bloodvessel or possibly of the surface of the serous membrane; this origin has very seldom been established; Miller and Wynn¹ have recorded an example. The histological appearances of primary malignant growths of the peritoneum vary very considerably; spindle-celled, round-celled, and irregular-celled, alveolar, fibrifying myxosarcoma and lymphosarcoma, endothelioma, and perithelioma (angiosarcoma) all occur. In some endotheliomas the microscopic appearances are confusing, in some parts resembling carcinoma, in others sarcoma. There may be considerable pigmentation from blood pigment in these tumors.

The shape of the tumors varies according to their position; retroperitoneal sarcoma and neoplasms originating between the layers of the great omentum are often flattened out so as to resemble a pancake. Retroperitoneal sarcomas may project forward into the peritoneal cavity and even become pedunculated. There may be multiple pedunculated growths without any manifest primary growth. A primary malignant growth of the peritoneum may be rounded or lobulated. The tumors may reach a considerable size. A primary malignant growth of the great omentum recorded by J. W. Ogle²

¹ *Journal of Pathology and Bacteriology*, Cambridge, 1908, xii, 267.

² *St. George's Hospital Reports*, 1867, ii, 350.

weighed thirty pounds. The origin of primary malignant neoplasms is more rarely intraperitoneal than retroperitoneal. This depends on the presence behind the peritoneum of structures, such as lymphatic glands, connective tissue, and the remains of the Wolffian body, Müllerian duct, etc., from which tumors may arise. It must, of course, be remembered that tumors manifestly arising in definite organs such as the kidneys and suprarenals, although retroperitoneal in position, are not included in this group of retroperitoneal sarcoma; as a rule, primary peritoneal and retroperitoneal tumors do not invade adjacent organs to any great degree. It may, however, be difficult to determine the exact origin of a malignant growth, and in some instances it must be left an open question whether the growth arose in an organ and spread out of it or whether it started independently of the viscus in question and subsequently infiltrated it. Thus a retroperitoneal tumor derived from the Wolffian body may be so intimately connected to the pancreas as to imitate tumors originating in that organ. In such cases the histological appearance of the tumor is the best guide to its origin.

The lymphatic glands and the connective tissues around the aorta and the periosteum of the vertebrae appear to be the commonest starting points of retroperitoneal sarcoma. A malignant cystic tumor resembling an ovarian cystadenoma, and probably derived from the remains of the Wolffian body, may occur. In rare instances a teratoma may become malignant. Montgomer¹ collected ten cases of this kind, and more recently Whitehead² recorded a malignant endothelioma of this origin.

Retroperitoneal sarcoma most often arises in the lumbar region and more frequently on the right side, but it may start in the middle line, in the iliac region, or in rare instances in the pelvis. There is frequently some local peritonitis with adhesions over the growth. The tumor is usually firm at first, but in a third of the cases it undergoes central necrosis and becomes cystic (Dutton Steele³). The contents may be hemorrhagic, myxomatous, or from infection purulent. The pseudocyst has in rare instances opened into the intestine or peritoneum. Thus in a case under my care, a cystic, spindle-celled sarcoma arising near the right kidney opened into the duodenum. The growth may exert pressure on the inferior vena cava, the spinal nerves, or on the abdominal viscera. The fixed parts of the colon are more liable to be narrowed by retroperitoneal growths. Tumors in the omentum, which may become cystic, may constrict the colon, and in the extremely rare cases of primary sarcoma of the gastrohepatic omentum, of which Cobb⁴ has been able to collect three examples only, the stomach may be depressed downward (A. P. Gould⁵). Secondary growths occur in 33 per cent. of the cases, and are found in the liver, lungs, and peritoneum, and in the latter position may give rise to ascites.

Etiology.—Sex apparently has no influence. In retroperitoneal sarcoma, according to Dutton Steele, the first, fourth, fifth, and sixth decades are most often affected, and 53 per cent. occur between thirty and sixty years of age. As in malignant disease elsewhere, injury has been thought to be a causal factor.

¹ *Journal of Experimental Medicine*, New York, 1898, iii, 259.

² *Ibid.*, 1901 to 1905, vi, 201.

³ *American Journal of the Medical Sciences*, Philadelphia, 1900, cxix, 311, and 1904, cxxvii, 939.

⁴ *Annals of Surgery*, 1906, xlv, 16.

⁵ *Medico-Chirurgical Transactions*, London, 1900, lxxxiii, 257.

Clinical Picture.—The clinical manifestations vary very much; most commonly there is a deep-seated abdominal tumor with somewhat variable gastro-intestinal symptoms, abdominal distention, and increasing weakness. Although usually fixed, the tumor is occasionally freely movable especially in the early stages. It may be regular and smooth, or nodular, more or less firm, or may show various degrees of consistency; it may receive transmitted pulsation from the aorta; it is usually not tender on palpation, and is more often to the right of the middle line; a growth in the omentum may resemble a spleen dislocated by extensive deformity of the spine. Primary growths, especially in the omentum, may induce ascites and so imitate tuberculous peritonitis, while a flat retroperitoneal sarcoma, although not palpable, may by pressure on the spinal nerves give rise to severe pain in the back imitating that of aneurism, caries, or malignant disease of the spine, and by compressing the inferior vena cava lead to œdema of the legs, genitals, and lower part of the abdomen with very noticeable enlargement of the subcutaneous abdominal veins. There may be profound anæmia before any tumor is palpable. On the other hand, malignant disease of the omentum may produce a large superficial mass forming a kind of shield in front of the intestines or may simulate enlargement of the liver.

The progressive growth of the tumor usually causes pain, and sometimes vomiting, constipation, and even intestinal obstruction. The sudden onset of abdominal pain, probably due to extension of the growth setting up local peritonitis, may be the first symptom. The growth may then be sufficiently large to be easily detected. Although the tumor may be of considerable size, the general state of nutrition is less affected than in malignant growths of the stomach or liver of the same dimensions. When retroperitoneal it may be resonant from the presence of intestines, colon, or stomach in front of it. When ascites is present the fluid may be, but is not necessarily, blood-stained. The fluid may be remarkably viscid from the presence of a "mucoid" body as in Miller and Wynn's case. The course of the disease is somewhat rapid, the average duration of retroperitoneal sarcoma from the onset of symptoms being eight and a half months (Dutton Steele). Death is usually due to exhaustion, but in a few instances has been sudden and due to rupture of the growth and hemorrhage into the peritoneal cavity.

Diagnosis.—From the variable character of the symptoms this is very difficult, and is usually arrived at only by a process of exclusion. Even although a peritoneal tumor be diagnosed, it is, except when there is definite evidence of other growths, impossible to be absolutely certain that it is malignant unless the abdomen has been opened. Even then removal of a piece of the growth for histological examination is necessary to be absolutely certain, for a considerable number of cases have been recorded in which the abdomen has been opened and closed on the discovery of an inoperable and apparently malignant tumor, and yet the tumor has subsequently disappeared (Greig Smith').

It is possible that these "vanishing tumors" (Power²) are, in spite of the absence of any naked-eye evidence, inflammatory in nature; in some instances a foreign body which has perforated the intestine may be the cause (Bland-Sutton³), and it is conceivable that in some the condition was due to actino-

¹ *Medico-Chirurgical Transactions*, London, 1894, lxxvii, 139.

² *Lancet*, 1899, i, 583.

³ *Ibid.*, 1903, ii, 1148.

mycosis. Bradford¹ has recorded cases in which, since tumors regarded as malignant disappeared and were succeeded by diabetes, the condition was thought to be due to pancreatitis. Malignant growths progress more rapidly, produce more marked constitutional symptoms, and give rise to pressure symptoms, such as pain, dilated abdominal veins, in a far greater degree than innocent tumors even though of great size. The differential diagnosis must be made from a number of conditions, such as pancreatic cysts, renal and suprarenal tumors, aneurism of the abdominal aorta which does not pulsate, retroperitoneal teratomas and innocent tumors, some cases of malignant disease of the colon with superadded suppuration, uterine fibromyomas, and when there is ascites from secondary malignant disease of the peritoneum and from tuberculous peritonitis. A horseshoe kidney, which is met with once in 1600 autopsies, has been mistaken for malignant disease. A nodular mass may resemble faecal accumulation, and enemas and purgatives may then be necessary to settle the diagnosis.

Prognosis.—In primary malignant disease of the peritoneum this is extremely grave. Operative interference is impracticable in many cases, especially in extensive retroperitoneal sarcomas. Tumors in the omentum or mesentery are more amenable to removal; thus, Pearce Gould removed a spindle-celled sarcoma, weighing twenty-one pounds, from the gastro-hepatic omentum of a man aged thirty eight years, who was in good health four years later. Removal of the tumor in an early stage, when it is more or less encapsulated, should lead to better results. In Williams² collection of 84 cases of retroperitoneal sarcoma, radical operations were performed in 12 cases, with recovery in 10. But exploratory laparotomy may show that the tumor is inoperable, from its wide extent, its close relation to the aorta or the inferior vena cava, and from the presence of secondary growths, or invasion of the viscera. Probably most of such cases are not recorded; this would appear to be a possible explanation of the remarkably good results tabulated by Williams.

Treatment.—Except when removal is possible, treatment is palliative and symptomatic. Opium and local applications are indicated for the pain, paracentesis to remove excessive ascites, and appropriate treatment for gastro-intestinal symptoms as they arise.

Secondary Malignant Disease of the Peritoneum.—Synonym: Malignant peritonitis. Secondary infection with carcinoma or sarcoma is far commoner than primary malignant disease of the peritoneum. That it occurs more frequently in women than in men is in part accounted for by influence exerted by malignant disease of the female sexual organs and of the mamma, while as a minor factor primary carcinoma of the gall-bladder, which is much commoner in the female sex, should be mentioned. It chiefly occurs in or after middle life.

Pathology.—Secondary malignant disease of the peritoneum usually spreads from a primary growth in the abdominal organs. Malignant infection may also extend into the abdomen from the chest, either through the lymphatics of the deep fascia of the parietes from the mamma, described by Handley³ as "epigastric invasion" of the peritoneum, or less commonly from the

¹ *Clinical Journal*, London, 1907, xxxi, 76.

² *American Journal of the Medical Sciences*, Philadelphia, 1903, cxxvi, 269.

³ *Lancet*, 1905, i, 1047.

cavity of the thorax as in primary carcinoma of the œsophagus. In primary malignant disease of the testes the lumbar glands may convey infection to the peritoneum. In some instances, as in generalized sarcoma, the infection is conveyed to the peritoneum by the blood stream. Primary malignant disease inside the abdomen may spread to some extent by the lymphatics and bloodvessels, or to a limited degree by direct contact; thus the omentum may become infected by a malignant growth in the pelvis even without the existence of any intervening adhesions. But the chief means of dissemination is the liberation into the peritoneal cavity of infective cells, which become widely scattered over the abdominal cavity and give rise to numerous metastatic growths, especially in Douglas' pouch.

A very curious condition, although it is not on a par with secondary malignant disease of the peritoneum, may be mentioned here. In papillomas, and more rarely in ruptured "dermoid" cysts (embryomas) of the ovaries, multiple growths of the same nature, due to implantation, may be found on the peritoneum. The process is not analogous to metastasis of malignant disease, for in the case of papillomas the peritoneal growths disappear when the original ovarian growth is removed. There is some reason to believe that innocent ovarian cystadenomas may occasionally behave in a similar fashion (Morton,¹ Bender²). When an ovarian cyst ruptures into the peritoneum, a condition spoken of as pseudomyxoma may result. It was regarded by Werth³ as the result of peritonitis, set up by the rupture, encysting the contents of the ruptured cyst. Myers⁴ has recorded a case in which this process appeared to have become malignant, as shown by invasion of the spleen and colon.

Morbid Anatomy.—The tumors may be so minute and discrete as to imitate tuberculous peritonitis, and the condition may then be spoken of as miliary carcinomatosis; in other instances the secondary nodules, being bigger and white in color, resemble caseous tubercles, while in some cases, especially of colloid carcinoma, the masses are of a very large size indeed. In women the ovaries may become markedly infiltrated with secondary growth, and as they may form tumors much larger than the primary growth, this sometimes suggests that the original tumor growth started in both ovaries. Thus in former times many patients from whom malignant tumors of both ovaries were removed, died with symptoms of intestinal obstruction; this was held to be due to recurrence of the growth, but was in reality due to the original although slow-growing tumor in the colon or stomach (Bland-Sutton⁵).

When the growths are miliary they are less translucent than miliary tubercles, when larger they are, as a rule, white, although they may be hemorrhagic or even melanotic, but they do not undergo caseation and softening. The growths, which are generally sessile or more rarely pedunculated, may be depressed in the centre or umbilicated. The whole of the peritoneum may be infected, but the omentum, the mesentery, and Douglas' pouch are the regions most frequently affected. Secondary growths may occur at the umbilicus, in the line of the round ligament, or, by spreading up the thoracic duct, give rise to enlargement of the glands above the left clavicle. There may, however, be most extensive malignant disease in the abdomen without

¹ *Practitioner*, 1904, lxxiii, 654.

² *Bull. Soc. anat.*, Paris, 1905, p. 480.

³ *Arch. f. Gyn.*, Berlin, 1884, xxiv, 103.

⁴ *Annals of Surgery*, 1907, xlv, 838.

⁵ *Lancet*, 1907, i, 1342.

any affection of the supraclavicular glands. When the growths are miliary the peritoneum may be otherwise normal, but in most cases the peritoneum shows chronic fibrotic changes (malignant peritonitis) resembling tuberculous peritonitis, the white nodules being surrounded with fibrotic thickening and often outlined by a rim of pigmentation. From the resulting contraction the omentum may become rolled up and the mesentery markedly shortened. The intestinal tract may be much shortened, the intestinal walls thickened, and the lumen narrowed. The diaphragm may be much thickened from growth, and in these cases miliary carcinomatosis of the pleuræ with effusion may result. When the funicular process of peritoneum is open, growths may be found in the tunica vaginalis testis. Adhesions may form and give rise to loculated ascites. In some instances evidence of acute or subacute peritonitis is found at the autopsy. From implication of the receptaculum chyli or of the thoracic duct the lacteals may be dilated with chyle, and from leakage true chylous ascites may result. A chyloform ascites due to fat globules is, however, commoner than true chylous ascites in malignant disease of the peritoneum.

Occasionally large masses of colloid growth are met with. More than one condition has been described as "colloid cancer" of the peritoneum; secondary growths due to a primary carcinoma of the stomach or colon are occasionally seen, but some of the cases formerly described as colloid cancer are probably primary retroperitoneal growths derived from the Wolffian bodies and myxosarcomatous or endotheliomatous in nature. The histological character of the tumors will, of course, vary with that of the primary growth, spheroidal-celled carcinoma being the commonest form.

Symptoms.—These come on gradually with loss of vigor, flesh, and appetite, are vague and, except that they point to the abdomen, somewhat indefinite. The symptoms vary since they are partly due to the primary growth, which is also usually in the abdominal cavity, and partly to the metastases in the peritoneum; in fact, it is usually impossible to assign the symptoms exclusively to one or other cause. Thus vomiting, dyspepsia, constipation, and some degree of abdominal pain might be due either to a primary growth of the alimentary canal or to the peritoneal metastases and resulting interference with intestinal peristalsis. In addition to the progressive wasting and weakness there may be varying degrees of abdominal tenderness. Pain, on the one hand, may be almost absent, or, on the other hand, there may be colic from obstruction to the lumen of the colon induced either by a primary carcinoma or by pressure or contraction exerted by secondary growths in the neighborhood. Tenderness and pain may be due to attacks of local peritonitis in connection with the growths.

Physical Signs.—In most cases there is ascites which may obscure the existence of any growths. The fluid may be clear, turbid, blood-stained, chylous, or chyloform (fatty or non-fatty). The abdomen may be much distended, the umbilicus everted, and the thorax encroached upon by the upward displacement of the diaphragm. The bases of the lungs may then be compressed, show signs of œdema, and induce orthopnoea. There may be distended veins under the skin of the abdominal wall as the result of obstruction to the flow of blood through the inferior vena cava, the veins run upward from the groin toward the axillæ, and do not converge toward the umbilicus, as is the case in obstruction of the portal vein. The umbilicus may be thickened from infiltration with secondary growth, and small nodules

may be felt in the line of the round ligament; it should be remembered, however, that small masses of fat in this position may imitate nodules of secondary growth. The skin of the abdomen loses its elasticity and, as the result of distention, shows lineæ albicantes. Even when not previously stretched the skin may from loss of its elasticity be thrown into numerous wrinkles and become somewhat harsh and rough to the touch. In a case without ascites, recently under my care, numerous and extensive lineæ albicantes appeared on the back in the last ten days of life.¹ Bedsores may occur in spite of skilled nursing.

Although there is usually emaciation, and to a greater degree than in primary malignant disease of the peritoneum, there is sometimes a very considerable amount of subcutaneous fat. A certain amount of dirty pigmentation of the skin is not uncommon; in very rare instances the skin of the abdomen shows a warty, pigmented condition called *acanthosis nigricans*. A certain amount of pigmentation of the face may be present. The appearance of multiple small angiomas in the skin has been regarded as a sign of malignant disease in the abdomen, but no stress can be laid on their presence, as they often occur in elderly people without any disease. Œdema of the feet, which may creep up the thighs onto the abdomen, may be due to pressure on the inferior vena cava, or in the later stages depend on cardiac weakness. In some cases definite tumors can be felt through the abdominal wall; very frequently the omentum is rolled up and palpable as a hard tumor or transverse ridge, which must not be mistaken for the lower margin of the liver. There may be friction over the tumors. Often when ascites is considerable no growths can be detected until the abdomen has been tapped, and when the growths are small nothing may be felt even then. There may be concomitant pleural effusion on one or both sides due to intrathoracic infection. The duration of the disease is seldom more than six months and is often much less.

Diagnosis.—When definite tumors can be felt in a patient who has malignant disease of the stomach, colon, etc., there can be no doubt. When tumors are palpable, but there is no history or definite evidence of malignant disease, the diagnosis must be made from tuberculous peritonitis, from faecal accumulation, and from the rather rare condition of multiple hydatids in the peritoneum. Tuberculous peritonitis is commoner in children and women, and may be accompanied by fever and by signs of suppuration about the umbilicus, while injection of the ascitic fluid into guinea-pigs would provide evidence of tuberculosis. Injection of tuberculin into the patient is another means of diagnosis. A blood-stained effusion is in favor of malignant disease. The discovery of multiple tumors in the abdomen should always suggest the possibility of faecal accumulation, and the effects of purgatives and repeated enemas on the position of the tumors should be tested. Multiple echinococcus cysts in the abdominal cavity run a slow course as contrasted with malignant disease, and may be definitely recognizable as cysts, while the history of a hydatid cyst in the liver makes the diagnosis of a similar condition in the peritoneum highly probable. In cases with marked ascites the presence or absence of tumors cannot be settled, and it may be impossible to decide whether the underlying condition is malignant disease or hepatic cirrhosis, simple chronic peritonitis, and other causes of

¹ *British Medical Journal*, 1908, i, 494

ascites, such as thrombosis of the portal vein, until the abdomen is tapped. It should be remembered that the malignant growths in the peritoneum may be too small to be felt. In these cases the presence of enlarged glands in the groins and above the left clavicle, and to a lesser degree a blood-stained, ascitic effusion, indicate malignant disease. Microscopic examination of the fluid withdrawn may, although it often does not, point definitely to malignant disease by showing the presence of small fragments of growth. The cytology of the effusion in malignant infection of the peritoneum, investigated by Dock,¹ Gwyn,² and others, may be of help in forming a diagnosis; large numbers of nuclear figures typical or atypical, and multi-nuclear endothelial cells are in favor of growth.

Prognosis is, of course, hopeless.

Treatment.—This is purely palliative and in the direction of the relief of pain by opium, morphine, and external applications to the abdomen, in the alleviation of gastro-intestinal symptoms, and the removal of ascitic fluid by tapping. To prevent re-accumulation of fluid after tapping, the injection of a dram of adrenalin (1 to 1000) diluted with water to half an ounce may be tried; it was shown by Meltzer and Auer³ that adrenalin retards both absorption and transudation.

¹ *American Journal of the Medical Sciences*, Philadelphia, 1897, cxiv, 655.

² *Ibid.*, 1904, cxxvii, 592.

³ *Transactions of the Association of American Physicians*, 1904, xviii, 207.

CHAPTER VIII.

SPLANCHNOPTOSIS. ENTEROPTOSIS. GLÉNARD'S DISEASE.

By THOMAS R. BROWN, M.D.

THE word enteroptosis, from the Greek words *ἐντερον* and *πτῶσις*, although in reality meaning a falling or descensus of the intestines, has been generally used to signify the descent of any or all of the abdominal viscera. The better word, however, is *splanchnoptosis*, while in describing the displacement of the individual organs special terms are used, such as *nephroptosis*, to signify descent of the kidney; *gastroptosis*, of the stomach; *spleno-ptosis*, of the spleen; *hepatoptosis*, of the liver; *coloptosis*, of the colon; while some of the more modern writers on the subject use the term *enteroptosis*, to mean descent of the intestines. The condition may be either partial or universal, and the different organs may be affected in varying degrees. It is also known as Glénard's disease, from the physician who first described it carefully, while some, as Kuttner, Dyer, Lockwood, Rose, and McPhedran, use the term *gastroptosis*, to mean prolapse of the stomach associated with the descensus of other viscera. It seems best, however, to use the word *splanchnoptosis* as the general term for the condition, and to confine the term *enteroptosis* to its correct meaning, that is, descent of the intestines. Etymologically speaking, however, all the terms are incorrect, and we should use *nephroptosis*, *gastroptosis*, etc., as A. Rose has especially insisted.

One thing must be remembered in the discussion of this disease, and that is, that in the earlier days *nephroptosis*, being much more easily recognized, was regarded as the most important feature, and to it were ascribed the protean symptoms often met with, while later investigations with better methods of diagnosis have shown that the displacement of the kidneys is usually associated with the displacement of the stomach, intestines, etc., and that many of the symptoms originally ascribed to floating kidney are in reality referable to displacement of other organs.

Historical.—We owe to the anatomists and pathologists the first recognition of *splanchnoptosis*. Morgagni was probably the first to describe it anatomically, while Virchow, in 1853, called attention to the displacement of the intestines, ascribing this to partial peritonitis, and regarding the mechanical effects of the ptosis as the cause and starting point of certain forms of dyspepsia and indigestion. Aberle gave the first clinical description of *splanchnoptosis*, while in several of the writings of the older clinicians, such as Rayer, Rollet, and Oppolzer, reference is made to the relationship between floating kidney and hysteria. Kussmaul first called attention to the clinical symptoms due to change in the form and position of the stomach, but there was no general interest in the subject of the displacement of the abdominal viscera until the work of Glénard, who

believed that in splanchnoptosis he had found the anatomical basis for one type of nervous dyspepsia. In an article appearing in 1885 he gave the results of his observations at Vichy, where many patients with digestive disorders flocked to take the waters, and ever since the appearance of his article the disease has been more and more widely recognized, until now it seems that perhaps the pendulum has swung too far, and too many disorders have been ascribed to it.

Anatomy and Embryology.—Before discussing the etiology of splanchnoptosis it will be advisable to call attention briefly to the anatomy and embryology of the abdominal organs. These viscera are held in their positions by a number of different forces: by the negative pressure of the thoracic cavity acting through the diaphragm, by ligamentous, vascular and peritoneal attachments, by the pressure of other abdominal organs, and by the supporting power of the abdominal muscles. Physiologically, no organ is absolutely fixed, but each is capable of a slight degree of movement, which may be caused by various physiological factors, such as the position of the patient, the amount of food ingested, the passage of urine and faeces, the respiratory and circulatory movements, and pregnancy. Thus we must not consider the abdomen to be filled with viscera that are normally fixed in their positions, but rather of organs capable of a slight degree of movement under a variety of conditions, the only fixed area in the abdomen being the *radix mesenterica*. As to the position of the various viscera but few words are necessary. The *liver* occupies the whole of the diaphragm on the right, as well as a part of the left side, and for the most part lies under cover of the ribs; its upper limits depend upon the position of the dome of the diaphragm, which, as Keith has shown, may vary between the third and sixth costal cartilages, although usually reaching to the level of the fifth chondrosternal articulation in the mammillary line. The *stomach* lies in the left hypochondriac and epigastric regions, its cardiac orifice being situated behind the seventh left costal cartilage, while the pyloric orifice is from three to four inches below the xiphisternal articulation in the median line when the stomach is contracted. The *transverse colon* passes across the upper part of the umbilical region, following closely the greater curvature of the stomach, which reaches to the infracostal line in the case of a moderate distention of that organ, while the splenic flexure reaches a higher level than the hepatic and is situated behind the stomach in the left hypochondriac region. The *kidneys* are situated at the back of the abdominal cavity, being lodged mainly in the epigastric and hypochondriac regions, that of the right side usually extending slightly into the umbilical and lumbar regions, that of the left side being from one-half to one inch higher, and rarely reaching below the infracostal plane. The *pancreas* lies over the first and second lumbar vertebrae from two and a half to five inches above the umbilicus, while the *spleen* lies very deeply, its long axis corresponding almost exactly with the line of the tenth rib, and its highest and lowest points being on a level with the ninth dorsal and first lumbar spines.

The *embryology* of the abdominal viscera is interesting because of the fact that a number of authors regard splanchnoptosis as a reversion of the abdominal organs to the embryonic type. Rosengart, from the study of a male foetus in the sixth month, found that the small curvature of the stomach extended perpendicularly up and down; the ascending and transverse colon extended in a straight line from the right lower inguinal region

diagonally upward; more than one-third of the right kidney lay upon the right iliac bone, while its upper end did not reach so high as the left. Upon the superior and exterior surfaces of the kidney the liver rested, while the lower portion of the kidney was pressed upon by the colon. This picture corresponds very closely to that given by Henle, in which there is a complete vertical position of the stomach. From this position of the viscera the position found in normal children and adults is produced by gradual ascensus of the various viscera, this being largely due to the fact that after respiration the liver rotates and rises into the dome of the diaphragm, and also undergoes a relative diminution in size.

Thus we see that in advanced cases of splachnoptosis the position of the abdominal organs very closely resembles that seen during embryonic life.

Pathogenesis.—As regards the pathogenesis of splachnoptosis divergent views are held, some believing that the condition is congenital, others that it is acquired, while others hold a middle ground. According to Glénard, the starting point of the condition is the falling of the right colic flexure, due to a weakening of the hepatocolic ligament. This may follow injuries, pregnancies, strains, dyspepsia, typhoid fever, appendicitis, or localized peritonitis, but is primarily due to a constitutional defect which is present in certain individuals as regards the strength and supporting power of the mesenteric tissues.

Stiller is another firm adherent of the congenital origin of splachnoptosis, and he believes that this inherited tendency is shown by a constant organic type, a slender skeleton, a long thorax, soft, flabby muscles, and a diminution of the panniculus adiposus, and that in these patients may be found a neurasthenic stigma, the floating tenth rib. According to him the basis of the disease rests upon an embryological defect, and the numerous causes which many authors give for this condition are in reality not primary causes, but simply factors which bring about a condition to which the body is already embryologically committed. Among the most important of these adjuvant causes Stiller especially mentions the rapid disappearance of fat due to various diseases, such as carcinoma and tuberculosis; he does not deny that an acquired splachnoptosis is occasionally possible, but these cases of movable spleen, kidney, or stomach of purely local nature are not associated with the subjective symptom-complex of splachnoptosis so rich in vague and indefinite symptoms.

Fuhs is also a firm believer in the costal stigma, and calls attention to the frequency of splachnoptosis among young unmarried Swedish girls who have never worn corsets and who have strong abdominal walls. The condition is also found frequently among Arab women, who also wear no constricting articles of clothing. Mathes believes that splachnoptosis is a constitutional and hereditary anomaly of the entire organism, a lack of vital energy of all the vital tissues, while Harris, who has paid especial attention to the etiology of nephroptosis, believes that this occurs in women with a particular body form, which can be expressed mathematically in terms of certain body diameters, and that practically all women with this body form have movable kidneys to a greater or less extent, while trauma is practically never the cause.

Landau believes that the primary cause is a weakness of the abdominal wall, in many cases congenital, while according to Bouveret, neurasthenia is primary, splachnoptosis secondary. Among other clinicians who believe

in the congenital origin of splanchnoptosis, in some if not all cases, may be mentioned Ewald, Lindner, Kuttner, Drummond, Schwerdt, Tuffier, and Montenuis. As already stated, Rosengart believes that in many cases there may be a real persistence of the fœtal condition. Riegel, on the other hand, is a firm believer in the acquired nature of the disease, and thinks that any condition which either increases the pressure above the diaphragm or diminishes the pressure below, may produce splanchnoptosis, such as pregnancy and parturition, wasting diseases, and the removal of abdominal tumors or ascitic fluid.

Many clinicians and investigators have laid especial stress upon the corset as the most important etiological factor, and among those who have especially called attention to this may be mentioned Einhorn, Dickinson, Kellogg, Bouveret, Montenuis, and Schwerdt.

Wolkow and Delitzen have carried out a number of interesting experiments in regard to the production of nephroptosis, and they conclude that the use of a belt may dislocate the kidney downward, may fix it, or may even move it upward according to the position of the belt, the high belt being obviously productive of the greatest harm; the corset, although it may fix the kidneys as long as it is laced, may increase their mobility by altering the form of the perivertebral spaces, thus acting as a recurrent trauma, and, indeed, the most plausible explanation of the influence of belt and corset is found in the action upon the shape of these spaces. According to these investigators, nephroptosis is an infallible index of an existing alteration of the intra-abdominal equilibrium. Janovski, from his experimental studies, thoroughly agrees with these conclusions, while Longyear believes that the ligamentous union of kidney and colon is an even more important factor in the etiology of nephroptosis.

Victor believes that the fundamental cause of splanchnoptosis is the inability of the abdominal walls to resist the weight of the viscera, that this is an inevitable accompaniment of the upright position, and that anything which increases the instability of the body will tend to produce this condition, such as marked straightening of the thorax at its lower extremity, marked anterior convexity of the lumbothoracic segment of the spine, and marked tilting forward of the pelvis.

Quincke gives three causes of splanchnoptosis: (1) Relaxation or stretching of the abdominal wall; (2) change in the form of the abdominal cavity; (3) stretching of one or more of the suspending ligaments; while Langerhans recognizes five causes: (1) Relaxation of the abdominal muscles; (2) hereditary predisposition; (3) pressure of clothing, as tight belts and corsets; (4) chlorosis; (5) nervous dyspepsia.

The most valuable contribution to the pathogenesis of splanchnoptosis is that of Keith, who believes that the condition is the result of a vitiated method of respiration. According to him the organs within the thoracic and abdominal cavities are poised between the muscles of inspiration and expiration, two contending sets, which throughout the life of the individual strive for the mastery. It sometimes happens that one organ is mainly affected, very frequently the kidney, sometimes the stomach, liver, or spleen; but even in such cases, although this organ may have suffered the chief attack of the inspiratory force, yet a full examination will show that all are displaced, although to a less degree. It must be remembered in this connection that all the abdominal viscera, except the kidneys, rest upon a visceral

shelf; thus, that of the liver is composed of stomach, duodenum, pancreas, colon, and right kidney; that of the stomach, of transverse colon, pancreas, duodenum, and left kidney; these shelves are maintained by the action and tone of the abdominal muscles. The reason that the right kidney is so much more frequently displaced than the left is because the liver acts directly on the latter, while in the case of the former there is a safety valve in the shape of the splenic flexure of the colon, which may enter or leave the hypochondrium with the greatest freedom; also the left kidney is bound to the spleen, and the fundus of the stomach represents a much lighter hammer than the liver. The adhesion of the base of the pericardium to the diaphragm, peculiar to man and the anthropoids, is a most effective aid against ptosis.

Briefly speaking, Keith's conclusions are as follows: (1) The contraction of the diaphragm is the factor which produces the displacement of the viscera in splachnoptosis, or Glénard's disease, and further, that of the various parts of the muscle, the crura are the most important agents in producing this result. (2) Before the displacement can be produced, either what he terms the thoracic supports of the diaphragm must have yielded, or the antagonistic abdominal muscles must have been hampered or weakened in their action, as, for example, by tight corsets. (3) The bonds which fix the viscera to the walls of the abdomen are of quite subsidiary importance. Displacement of liver and stomach arises chiefly from two causes, relaxation or paresis of the abdominal muscles, which maintain the visceral shelves, or, more frequently, constriction of the body cavity by clothing or disease so that the normal respiratory swing forward cannot take place.

Thus the theories regarding the pathogenesis of splachnoptosis may be broadly placed in three groups: (1) The condition is congenital, and is due to an inherent weakness of the tissues which support the abdominal viscera; such factors as clothing, trauma, disease, etc., are not the primary causes, but simply contributory ones, which bring out or accentuate the inherited tendencies. (2) The condition is due to the pressure of clothing, trauma, weakness of the abdominal walls, various diseases associated with marked loss of strength or weight, various spinal and thoracic diseases, pregnancy and parturition, abdominal tumors, ascites; in other words, a number of conditions, each of which has as its result the weakening of the various visceral supports, due in some cases to pressure, in other cases to adhesions, in other cases to trauma, and in still other cases to a marked diminution of the supporting power of the abdominal wall. (3) That each of the foregoing may be the primary cause of the condition; in other words, that there may be both an acquired and a congenital form of splachnoptosis. As to the method of the production of the various ptoses, the views of Keith are of great interest.

The writer has been studying splachnoptosis most carefully during the past eight years, and the more it is studied the more one is compelled to believe in the extreme importance of a definite congenital defect as the underlying cause in very many patients. This latent predisposition is transformed into the actual disease by some of the factors mentioned above; there are, however, instances of acquired splachnoptosis with no evidence of the characteristic body form or other constitutional defects peculiar to most of these patients. This suggests that while in the majority of instances the condition is due to a congenital defect, yet in some it may definitely be of the acquired type.

Frequency.—In discussing the frequency of splanchnoptosis we will naturally and necessarily quote from the tables of the frequency of displaced kidney, displaced stomach, displaced liver, etc., because in the vast majority of cases the descensus of one organ is associated with that of other organs as well, although for various reasons the one organ alone has been considered. Naturally the only tables of real value in this connection are those of recent years, when the condition has been well recognized, and when clinicians have been on the alert to diagnose it. Lawson Tait stated that there were no movable kidneys, and yet we have but to look at many of the older pictures, notably Botticelli's women and Menling's Eve, to realize that the condition has unquestionably been present for a long time, although it is highly probable that it is on the increase. The condition has been met with in practically all the races in which it has been carefully looked for, and cases have been described quite frequently among the Arab women and other tribes among whom tight clothing is unknown.

Glénard reported 148 cases among 1310 patients at Vichy; Stiller meets from 300 to 400 cases of splanchnoptosis in his private practice every year; while Kuttner found 100 cases of this condition among 4000 patients in Ewald's clinic; Kuttner also states that he frequently found floating kidneys in children, which is also the writer's experience and that of Blum. Einhorn in one year saw 70 cases of splanchnoptosis among 1080 males, and 277 cases among 832 females. Of 240 cases of enteroptosis proper, 20 were in men and 220 in women, and in these cases movable kidney was met in 212, movable liver in 23 cases in association with movable kidney, and in 12 patients without movable kidney. Enteroptosis alone was met with in 15 patients, 1 male and 14 females, and movable kidney alone 57 times, 21 males and 36 females. Thus in Einhorn's practice, which is largely devoted to digestive disorders, and in which, therefore, the figures are extremely high, ptoses of the abdominal viscera are met with in 6.5 per cent. of the males and 33.25 per cent. of the females. Arneill found records of splanchnoptosis in 11 men and 69 women of 2004 cases of which he had records since 1892, but these figures are undoubtedly too low.

As regards the incidence of *movable kidney*, many large series of cases have been reported, as this displacement has been studied far more than that of any others of the abdominal viscera. The older anatomists and clinicians noted it but rarely; thus, Ebstein found movable kidney only 5 times in 3658 postmortems, while Landau reports only 4 cases among 6999 hospital patients.

The reports of the past few years, however, show the great incidence of this condition, especially among women; thus, Lindner found that one out of every four women had displaced kidney; Mathieu found 85 cases among 306 patients; Larrabee reports 112 cases among 272 patients in the Out-patient Department of the Boston City Hospital; Glénard, in his statistics published in 1893, found 481 cases of floating kidney among 3788 patients, 2.7 per cent. in men and 22 per cent. in women; Wolkow and Delitzen found 66 cases among 221 women; Einhorn found nephroptosis in 1.81 per cent. of males and 20.6 per cent. of females. According to Burnam, who has carefully investigated the records of the Johns Hopkins Hospital, one out of every 5 women had a movable kidney, and one out of every 50 men, these figures agreeing absolutely with those of Tuffier. In Kuttner's series of 667 cases collected from the literature and from his own practice there

were 584 women and 83 men. McPhedran and Stiller are the only two authors who deny the much greater frequency of nephroptosis in women.

As regards the grade of displacement, Glénard describes four degrees: (1) When the lower portion of the kidney can be felt only during deep inspiration; (2) when the greater portion of the kidney can be felt, but the upper border cannot be made out; (3) when the whole kidney can be palpated during deep expiration; and (4) when the entire kidney can be palpated, and is capable of being moved about the abdominal cavity, this latter being the true floating kidney. According to Ewald and Kuttner, the degrees of displacement are: (1) The kidney shows a demonstrable respiratory movement without being dislocated to any extent; (2) the kidney shows a dislocation of the first degree, that is, we are able to palpate from one-third to two-thirds of the organ, which can be moved by means of the hand, and is more or less dislocated forward; (3) the kidney shows a dislocation of the second degree, that is, is palpable over its entire extent; is easily moved by the hands and during respiration, and lies near the anterior abdominal wall, or, at least, is easily brought there; and (4) the kidney is dislocated and fixed in an abnormal position. In Kuttner's and Ewald's series of 79 cases associated either with dilatation or displacement of the stomach, in 47 the right kidney was displaced, in 7 the left kidney, and in 25 both kidneys; 18 cases fell under the first grouping, 64 under the second, 19 under the third, and 3 under the fourth.

As regards *age*, displaced kidney, as well as ptoses of the other viscera, is frequently met with in very young children, in several cases under three years of age. In Kuttner's series of 326 cases, 6 were found between one and ten years, 32 between ten and twenty, 82 between twenty and thirty, 123 between thirty and forty, 49 between forty and fifty, 26 between fifty and sixty, and 8 between sixty and seventy; that is, the great majority of these patients when seen by the physician for the first time are between twenty and fifty years of age. Larrabee reports 5 cases over sixty years and one over seventy. Blum has recorded many cases among children, and we have noted the condition frequently in very young infants.

As to *race*, in Larrabee's series of 112 cases among 273 patients, of 93 Americans nephroptosis was found in 37 per cent., of 69 Irish in 51 per cent., of 41 British in 49 per cent., and of 22 Russian Jews in 30 per cent.

As to the relation of pregnancy to floating kidney, in Kuttner's series of 94, 40 patients had never borne children, 10 had borne one child each, and 44 had borne two or more children each. In Landau's series, 31 of 34, on whom notes were made, had borne no children, while in Lindner's series of 75 patients, 24 had never been pregnant, 12 had been pregnant once, 30 had been pregnant two or more times, while of the others no notes were to be found. Of Drummond's 27 women patients 16 had borne children, while of Larrabee's 112 cases, 51 were nulliparous and 50 multiparous.

As regards the frequency with which right, left, and both kidneys are affected, in Kuttner's series of 727 cases, in 553 the right kidney alone was movable, in 81 the left kidney alone, and in 93 both kidneys. In Einhorn's series the right kidney alone was movable in 77.3 per cent. of the cases, the left kidney alone in 2.1 per cent., and both kidneys in 20.6 per cent.

As regards the incidence of *gastroptosis*, figures are of little value because, in the first place, the condition is generally overlooked unless associated with gastrectasis or unless special examination is made either by inflation

or the use of the x -rays, and in the second place because many of the symptoms, which in reality were due to a displacement of the stomach, have been ascribed to displacement of the kidney and treated accordingly. Francine has collected 100 cases, and Kuttner reports 15 cases of marked gastropotosis, in only 2 of which the abdomen was pendulous, in 4 the abdominal muscles were of normal tone, while in 9 they were soft and flabby. Kuttner and Ewald report 100 cases of displaced kidney, 94 in women, 6 in men, in 79 of which there was either a dilatation or displacement of the stomach. Smithwick, in 68 cases of splanchnoptosis, found the right kidney affected in all cases, the left kidney in 20 per cent. of the cases, the stomach in 62 per cent., and the liver in 7 per cent. The writer's experience has been that in a large proportion of the cases of splanchnoptosis the stomach is involved to a greater or less extent.

Glénard has reported 51 cases of *displaced liver*, 32 being associated with movable kidney, and of these, 30 were women and 2 men. Einhorn reports 21 cases of displaced liver in 369 women, 9 cases in 439 men; a moderate degree of abdominal flaccidity was present in 16 of the 21 women. Graham has collected 68 cases of displaced liver, 55 in women, 13 in men, and Savelieff has recently collected 118 cases from the literature. Clarke and Dolley report a case of congenital floating liver with a definite mesohepar.

Several isolated cases of *floating spleen* have been reported, although, unless the spleen is markedly displaced, a ptosis is difficult to determine, because of its position, which can only be determined by the most careful percussion and because of the absence of symptoms. Of Einhorn's 1912 patients, in which there were 347 cases of splanchnoptosis, ptosis of the spleen is mentioned but once. The frequency of displacement of the pancreas, of the colon, and of the sigmoid flexure is difficult to determine. The displacement of the pancreas is of interest because Glénard's *corde colique transverse*, which he believed to be the constricted transverse colon, Ewald regards as the descended pancreas.

Cardioptosis, or the descensus of the heart, has been described in a few cases. Einhorn found it 22 times in 926 patients, 18 men, 4 women, and in about one-half of these cases there was an associated hepatoptosis. He thinks the condition is probably due to the downward displacement of the diaphragm.

The association of displacements of the pelvic organs with splanchnoptosis is a subject to which very little attention has been paid, and yet it is one of extreme importance, because retroflexion of the uterus is a very frequent accompaniment of displacement of various abdominal organs. Smithwick found that of 34 women with splanchnoptosis, 80 per cent. had retroversion of the uterus generally in a marked degree, and Smith believes that the majority of patients coming to the gynecologist have splanchnoptosis.

Symptoms.—We will discuss the symptomatology of splanchnoptosis in general, and later call attention to the symptoms especially referable to each of the displaced organs. In the first place, it must be insisted upon that there may be absolutely no symptoms whatsoever, and, in the second place, that the intensity of the general symptoms does not, as a rule, depend so much upon the extent of the displacement of the various viscera as upon the degree the nervous system is involved. In this connection we must not forget that all of these symptoms have been ascribed in turn to each of

the viscera, according to whether kidney, stomach, liver, or intestines were the special object of study, and yet that in the vast majority the symptoms are referable to a displacement of all or most of the viscera, although the degree of displacement may be different. The usual picture of a case which presents symptoms is that of a thin, pale, young woman or man of slight build with dyspeptic and nervous complaints, a nervous expression, and thin, soft abdominal walls in which there is a marked lack of tone. Some have called attention especially to the juvenile expression of the face, others to the fact that the picture closely resembles that met with in phthisis. We may have symptoms of the most aggravated type of neurasthenia, to which are added various abdominal symptoms, such as indigestion, constipation, pain in various portions of the abdomen, a feeling of lack of abdominal support, frequent, sometimes painful urination, backache, sideache, various respiratory symptoms, cardialgia, pain during or after meals, sometimes nausea and vomiting. All of these symptoms are aggravated by standing, walking, or exercise, while the prone position causes them to markedly diminish and sometimes to disappear for the time being. It is also noteworthy that the symptoms become less if the abdomen is lifted up either by an artificial support or by the hands of the physician as he stands behind the patient—the so-called “belt test” of Glénard. The greater the congenital anomaly, and the earlier the adjuvant factors act upon one so predisposed, the sooner will these symptoms make their appearance. Among the more important objective symptoms may be mentioned a flattened abdomen, often with hypoplastic walls, a bent spinal column, sometimes an abdomen flattened in the epigastric region but distended below the level of the umbilicus, sensitiveness to pressure near the tenth rib in some cases, a cord-like transverse colon (or displaced pancreas, according to Ewald), through which the pulsation of the aorta can be distinctly felt, while in some cases visible swellings can be seen in abnormal locations due to displacement of the various viscera.

Glénard divided the symptoms into three special groups: hypostasis, or lack of tone of the abdominal walls, splachnoptosis, and enterostenosis. The first shows three special symptoms: deformity of the abdomen, flabbiness of the abdominal walls, and the ease with which the hypochondrium can be compressed. Splachnoptosis shows four special signs: splashing sounds in the stomach, epigastric pulsation, floating kidney, and ptosis of the transverse colon. Enterostenosis has three special signs: a palpable contracted band of the transverse colon, of the cæcum, and of the sigmoid flexure. The three symptoms on which Glénard put especial stress are a stenosed and displaced colon, movable kidney, and deformed liver; he believes that the starting point of the condition is a falling of the right colic flexure due to a weakening of the hepatocolic ligament, which may follow injury, pregnancies, strains, wasting diseases, etc., but which is primarily due to a constitutional defect.

Among other symptoms which have been described may be mentioned pain and fatigue in the early afternoon and in the very early hours of the morning, these hours, according to some, corresponding to the period of duodenal digestion, marked nervous crises, hysteria, insomnia, despondency, general malaise, loss of memory and of the power of concentrating attention, and irritability.

Meinert believes that chlorosis in young girls is sometimes a crisis of

splanchnoptosis, while Quincke lays especial stress upon the respiratory symptoms, due in part to the lack of tone of the abdominal muscles and partly to the deformity of the lower portion of the thorax, which disturbs the relation between the diaphragm and ribs in many ways; due to these causes we may find shortness of breath on standing or exertion, cardiac oppression or distress, and sometimes dizziness or weakness, possibly due to vascular changes, possibly to stretching of the splanchnic nerves.

Glénard himself describes four forms of splanchnoptosis, the gastro-intestinal, the lithæmic or hepatic, the neurasthenic, and the cachectic, and believes that in many cases the patient goes from one form into the other in the order named. Keith, who has paid so much attention to the respiratory phenomena of splanchnoptosis, calls especial attention to the frequency with which cervical curvature or ewe-neck is met. He believes that from a clinical point of view the most important of the visceral bonds is the gastrohepatic omentum, and that more than half the results of ptosis of the liver and stomach are due to compression, straining or distortion of the bile, blood, nerve and lymph channels contained in this omentum. He believes that gallstones are commonly present in cases of splanchnoptosis, explaining this by the descent of the duodenum, head of the pancreas, and termination of the common bile duct, which is invariably caused by the displacement of the liver, and which makes the entrance and exit of the bile to and from the gall-bladder extremely difficult.

As to the relationship between neurasthenia and splanchnoptosis, there is a marked difference in opinion, some holding that neurasthenia is primary and enteroptosis is secondary, others the reverse, while a third group holds, and probably rightly, that in most cases each represents a congenital fragility of tissue independent primarily of each other, but frequently associated and reacting very deleteriously upon each other.

It must strike our gynæcological brethren that the symptoms which they have ascribed to retroflexion of the uterus bear a striking resemblance to those of splanchnoptosis, and this accentuates the fact already noted that in the vast majority of cases retroposition and retroflexion of the uterus are but associated signs of a general splanchnoptosis, and to expect relief by a ventral fixation of the uterus seems chimerical, to say the least.

Gastroptosis.—Gastroptosis, although one of the less frequently recognized of the visceral ptoses, is one of the most important as regards symptomatology; undoubtedly many of the symptoms referred to displacement of kidney or liver are in reality due to an unrecognized displacement of the stomach. We have already referred to the vertical position of the stomach in embryonic life, and to the two views regarding the etiology of this condition, that is, that the condition may be congenital or may be acquired. As to its frequency, it is being more and more recognized as it is being more carefully investigated; thus, Meinert finds displacement of the stomach in 80 per cent. of the patients in his gynæcological clinic, while Thompson, who has noted many cases, finds it six times more common in women than in men.

In the downward dislocation of the stomach we have two main forms: First, the whole stomach may be dislocated downward, and second, the stomach may occupy a more vertical position, although, strictly speaking, the former is rare, due to the firm fixation of the cardiac end. The most frequent form is the subvertical, the next is the vertical, while the loop,

erescens, or garland form is less common. The vertical or subvertical forms are peculiarly liable to dilatation, this being, of course, aided by a general weakness of the patient, frequent overloading of the stomach, relaxation of the abdominal wall, pregnancy and parturition, rapid loss of weight, and constriction due to corsets. As regards symptoms, there may be none whatsoever; in fact, the symptoms directly referable to the stomach are largely dependent upon the associated dilatation or the motor or secretory anomalies. Steele and Francine found some dilatation of the pyloric extremity in each of 70 patients examined by them. We must, therefore, regard the symptoms met with in gastropotosis as largely due either to a perversion of the motor or the secretory powers of the stomach, or to a dilatation with its characteristic symptoms due to the retention and decomposition of food, and the associated auto-intoxication. If the ptosis is marked, it is hard to see how it is possible to prevent difficulty in the propulsion of food, leading to increased fermentation in the stomach and subsequent distention and atony. So long as the stomach is not overtaxed there is little or no distress, for if the ptosis is not great the stomach is easily able to perform the additional amount of labor. If, on the other hand, excessive work is thrown on the stomach, as after very large meals, rapid eating, the ingestion of much easily fermenting food, or the upright position immediately after eating, the stomach is unable to do the excess of work, and dilatation occurs.

The symptoms which are especially referable to the displacement of the stomach are distress and flatulence after meals, sometimes nausea and vomiting, tachycardia, anorexia, coated tongue, numbness and dizziness, pain, which may be due either to fermentation, associated catarrh, increased acidity, or the pressure of the displaced stomach on the solar plexus, and a feeling of lack of support, while in some cases the dread of gastric disturbances leads to slow starvation and cachexia. Many of these symptoms are, of course, in the main due to the associated dilatation or motor insufficiency, while in all cases the quantity of food seems to be of more importance than its quality in the production of symptoms. Rose believes that the condition is identical with atonia gastrica. Whether the dilatation is due to gastropotosis alone, or whether a displaced kidney plays a part in its etiology, will be discussed subsequently. A splashing or a rumbling sound in the stomach is frequently met with. Of course, it must be remembered that in many cases the symptoms are more referable to the neurasthenia so frequently associated with this condition than to the displacement of the organ *per se*. In gastropotosis, as in the other ptoses, the symptoms are usually lessened by the prone, and increased by the upright position.

As to the effect of ptosis upon the gastric secretion subacidity is the rule. In 20 patients examined by the writer in this connection, only one showed a hyperacidity; there was a slight diminution in the free hydrochloric acid, with no or slight dilatation, while as the dilatation became more marked the free hydrochloric acid diminished, and was absent in most of the patients with very high grades of dilatation, thus suggesting that the subsequent dilatation is of more importance than the primary ptosis as regards the gastric secretions. Steele and Francine, from a study of 70 cases, also found that diminution of free hydrochloric acid was the rule.

Nephroptosis.—Of all the divisions of splanchnoptosis, nephroptosis or displaced kidney has absorbed by far the greatest amount of attention and

study, an unfortunate condition in many ways, because to it have been ascribed so many of the symptoms which undoubtedly were referable to the displacement of other organs that investigation of the other ptoses has lagged far behind, and yet in the vast majority of cases nephroptosis is but a part of a general splanchnoptosis.

Some, such as Israel, Litten, Morris, and Kuttner, believe that the so-called first degree of displacement of the kidney is not pathological. Harris, who is a firm believer in the congenital origin of nephroptosis, and who found a movable tenth rib in 61 of 110 cases, believes that the characteristic sign is a peculiar body form, a marked contraction of the lower end of the middle zone of the body, with a diminution of the capacity of this portion of the body cavity. Becker and Lenhoff divide the distance from the supra-sternal notch to the upper edge of the symphysis pubis by the least circumference of the abdomen, for the sake of convenience multiplying this by 100; the average index was 77, and they found that if it was above 77 the kidneys could usually be felt, while if below 75, as a rule they could not be felt. These views are in harmony with the congenital theory of nephroptosis and are also borne out by numerous clinical facts. Thus Albarran, Sheldon and others have noted numerous cases in the same family, while many cases have been reported in very young children, in some cases under two years of age, where none of the ordinary predisposing causes had been met with.

As we have said before, many of the symptoms which have been referred to nephroptosis are in reality referable to a displacement of the stomach and intestines. Among symptoms definitely referable to the kidneys themselves may be mentioned a feeling of lack of support in the back and flank, frequent urination, associated with burning sensations, which is sometimes diagnosed and treated as cystitis, pain which may be either continuous or intermittent, and which is often relieved by the patient lying down, symptoms very suggestive of appendicitis or gallstones, pain, jaundice, etc., which may be due to pressure, and which, according to Keith, is very common, and may even lead to the formation of gallstones. There may be intermittent hydronephrosis with polyuria after the reduction of the kidney to its normal position, the so-called Dietl's crises, consisting of paroxysmal attacks of intense pain, nausea, and vomiting, which some believe due to intermittent hydronephrosis, while others consider that they are caused by torsion of the renal vessels and nerves. Other symptoms are suggestive of gastric ulcer; blood, albumin, and casts may occur in the urine, which, however, may be referable to the trauma of the kidneys produced by palpation; gangrene of the kidney is an extremely uncommon condition, while some believe that the displaced kidney produces and maintains a congestion of the pelvic organs.

Ruggi believes that some of the neurasthenic symptoms met with in nephroptosis are due to a perversion of the internal secretion of the kidneys, a view, however, which at present has no basis in fact. Some patients complain of feeling a moving mass in the abdomen, others have definite symptoms of sciatica and crural and intercostal neuralgia. Intermittent hæmaturia and nephritis have been described, while in two cases of post-operative pyelitis examined by the writer the fact that the affected kidney was in each case the displaced organ suggests that its resistance is lowered by the displacement.

Burnam, from his most interesting study of the cases of nephroptosis at the Johns Hopkins Hospital, finds that only one case in ten of movable kidney has marked local symptoms, the cardinal symptoms being pain in the side, gastric symptoms, and nervousness. The dragging dull pain in the flank is about three times as common as acute attacks of Dietl's crises, while cases of Dietl's crises are commoner than those which have both the dragging pain and crises. About one out of every eight cases with Dietl's crises shows a beginning hydronephrosis, about one out of every three cases shows marked gastric symptoms, and one out of every three cases shows pronounced nervous symptoms. Tuffier calls attention to the fact that although traumatism rarely is the cause of renal displacement, it often calls attention to an organ already displaced by calling forth active symptoms. He believes the gastric symptoms in nephroptosis are due to an atonic gastric dilatation, while of the paroxysmal pains in the renal region he describes two kinds: first, slight pain followed by the passage of much clear urine, and second, the pain associated with intermittent hydronephrosis, and suggestive of renal calculus, there usually being a renal tumor to be made out during the paroxysm, while the amount of urine, which is markedly diminished with the onset of the pain, is often markedly increased with its subsidence, part of this increased flow being due to a reflex action of the healthy organ. Kelly, by catheterization of movable kidneys, has shown that a slight hydronephrosis is the rule, for he found the average capacity of the renal pelvis in 65 cases of nephroptosis to be between 12 and 13 cc., the normal capacity being 7 to 8 cc., while Tuffier divides the cases of nephroptosis into the simple and the hydro-nephrotic.

The relationship between nephroptosis and dilatation of the stomach has not as yet been definitely determined. According to some the gastrectasia is definitely secondary to gastroptosis, while according to others the displaced kidney plays a very important role in the production of the dilatation. Bramwell reports a case of movable kidney producing pyloric stenosis and constriction of the duodenum. Müller-Warneck believes that the right kidney when movable falls inward and forward, producing pressure upon the descending portion of the duodenum, which in turn produces dilatation of the stomach by the constricting bands caused by the ptosis, while Bartels believes that it is the kidney itself that compresses the duodenum, a view concurred in by Malbranc, Schütz, and von Fischer-Benson. Others, however, believe that floating kidneys play no part in the production of gastrectasia, among whom may be mentioned Oser, Leube, Ewald, Nothnagel, and Lindner. Drummond found no cases of dilated stomach in his 31 cases of movable kidney; Kuttner has found gastrectasia associated with movable left kidney, which is hard to explain on Müller-Warneck's theory, and he found no improvement in stomach symptoms after fixation of the kidney; Litten believes that in some cases gastrectasia may cause floating kidney, while Lenhartz found no cases of movable kidney in 16 cases of gastrectasia. It seems far more probable that the dilatation of the stomach so frequently met with in splanchnoptosis is in the vast majority of cases etiologically dependent upon the gastroptosis, and that there is no causal relationship between it and floating kidney except in very rare instances. There is no question but that chronic appendicitis is frequently associated with displaced right kidney, and this fact should always be kept

in mind; it is often advisable to remove the appendix at the same time that the kidney is stitched into position. Many have also called attention to the relationship between floating kidney and obstruction of the common bile duct.

Hepatoptosis.—Floating liver is frequently not diagnosed, due partly to the difficulty in making out a moderate degree of displacement, partly because it is frequently regarded as an enlarged liver and partly because by the vast majority of observers the kidney is the only organ carefully investigated in splanchnoptosis; yet the tables of Graham, Einhorn, Landau, and others show that the condition is not rare. Floating liver was first noted on the dissecting table by Sauvage and Portal, and in 1866 Cantani described the first case in a living patient. The symptoms of the condition are rather indefinite. There may be spontaneous pain, which is sometimes brought on by jumping, walking, raising the right arm, sneezing, coughing, and yawning, while sometimes paroxysms of pain occur without apparent cause; in the writer's experience pain has been a very common symptom, usually relieved by making the patient lie on the back or on the right side, or by manual replacement of the organ. The pain is commonest in the right hypochondriac and epigastric regions, radiating thence toward the shoulder or the flank. Pressure, although rarely painful, often produces peculiar sensations in various portions of the body, especially the arms and shoulders.

Many patients complain of digestive symptoms, meteorism, constipation, or of feeling a movable body, a symptom frequently met with in nephroptosis; others of bearing-down sensations and colicky pains, while among other symptoms which have been described in occasional cases may be mentioned respiratory disturbances, palpitation, ascites, hemorrhoids, œdema of the lower extremities, albuminuria, polyuria, purpura, jaundice, and recurring hemorrhage from the stomach. Keith has especially insisted upon the important symptoms referable to hepatoptosis, believing that they are caused by the compression of the bile, blood, nerve and lymph channels contained in the gastrohepatic omentum; he believes that gallstones are very common. Steele has shown by experiments on the cadaver that biliary obstruction is met with in floating liver, pressure in the common duct being between two and three times the normal. Landau describes three grades of hepatoptosis: (1) moderate descent with anteversion or retroversion; (2) marked descent either with lateral displacement toward the right, or with anteversion or retroversion; (3) vertical or oblique displacement, the left lobe descending into the abdominal cavity.

Descent of the Intestines.—This is practically met with in every case of splanchnoptosis, but the difficulty of making a definite diagnosis has made most observers minimize its importance; yet the intestinal symptoms are many and diversified, and among them may be mentioned constipation, fermentative dyspepsia, mucous colitis, which is quite common, various symptoms due to stenosis of the intestines, marked fulness in the lower half of the abdomen, peristaltic movements in the displaced intestines, often made out if the abdominal wall is thin, and pain and fatigue in the hours corresponding to the period of duodenal digestion, which O'Connell thinks are three in the afternoon and two in the morning in the majority of cases. Ewald believes that the intestinal disturbances are due to the dragging of the bands on which the sections of intestines are hung, this producing reflex irritation of the intestines, disturbances in their circulation, their motor and

secretory functions, while, due to the sinking of the transverse colon, the horizontal portion of the duodenum is pulled downward. The formation of adhesions probably plays a part in the production of these symptoms. The small intestines, containing liquid fæces, are but slightly affected by ptosis, but in the large intestines, containing solid fæces, a loop or kink is of much more serious importance and may produce marked constipation. Recent work has shown that ptosis and kinking of the sigmoid flexure is an extremely important factor in this connection, while the transverse colon, which in its ptosis assumes an M or V shape, often pulls down the stomach in its descent.

Descent of Spleen and Pancreas.—The descent of the spleen has been rarely described, because it is difficult to recognize slight displacements and because symptoms are usually absent. In a few cases which have symptoms the patients complain of dragging, uneasy sensations in the back and side. All grades of splenic displacement have been described, while the fact that several cases have been found in members of the same family suggests a congenital origin. The organ is usually enlarged, while sometimes torsion of the pedicle may occur with gangrene and the need of immediate operation to preserve life.

As regards the pancreas, we must remember that a segment of this organ is firmly bound to the radix mesenterica, and is, therefore, fixed, but Ewald believes that the other portion may undergo descensus, and may be felt as a constricted cord, which Glénard, however, believed to be the transverse colon.

Diagnosis.—The diagnosis of splachnoptosis should be easily made by all careful observers. The diagnosis can often be made when the patient is first seen; the characteristic body form, the expression frequently described as juvenile, the evident neurasthenic tendency, so frequently associated with splachnoptosis, should at once call our attention to the possibility of this condition. Harris, Becker, Lenhoff, and others have expressed this peculiar body form mathematically, and the measurements which can be made in a few moments are often very suggestive. Others insist on the importance of a floating tenth rib in this condition. In all cases the vague and protean symptom-complex of nervous and digestive disorders, inability to do hard mental or physical work, and the other symptoms which have been described should make us suspect the condition. We must remember, however, that many patients show no symptoms whatsoever, and in such the diagnosis can only be made by a careful physical examination.

As regards the diagnosis of *gastroptosis*, splashing or succussion sounds are frequently heard, there is commonly a sinking in of the epigastrium, with a bulging in the region of the umbilicus or below it, while if the abdominal wall is thin peristaltic waves may be definitely seen. To exactly diagnose the form and position of the stomach we may attempt to percuss the organ, use auscultatory percussion, or we may with the stethoscope over the stomach determine the length of time it takes for fluid to pass from the mouth into this organ. All these methods, however, are inexact, and it is therefore advisable either to distend the stomach with carbon dioxide gas, making the patient take bicarbonate of soda and tartaric acid in separate glasses; distend by air through a stomach tube, according to Runeberg, which has the added advantage of telling us something regarding the tone of the stomach wall; make use of Türk's gyromele; Einhorn's method of

gastric diaphany, an electric light being introduced into the stomach; or inflate the stomach with water. Recently much work has been done, notably by Wordew, Sailer, Pancoast, and Davis, in using the *x*-rays in diagnosing this condition. Bismuth subnitrate, suspended in mucilage of acacia, two ounces to the pint, is introduced into the stomach, and immediately afterward an *x*-ray photograph taken, the breath being held in full but not forced inspiration and the patient standing. The exposure does not last more than fifteen seconds, the plate being in contact with the anterior wall and the rays thrown posteriorly. Siphoning off the bismuth emulsion immediately afterward is advisable to prevent toxic symptoms. If by any of these means the greater curvature is found at or below the umbilicus, and the pylorus and lesser curvature displaced downward, the diagnosis can be made.

In *nephroptosis* sometimes the displaced kidney can be distinctly seen through the thin abdominal wall, while in many cases a sinking in of the flanks can be noted. The only satisfactory way to make the diagnosis, however, is by means of bimanual examination and palpation of the organ. The patient should be examined both in the standing position, lying on the back, and lying on either side, while it is extremely important in many cases to make the patient walk around, hop, jump, or support herself with both hands on the side of the bed before making the examination. Many mistakes have been made by examining patients in bed after a night's rest when the kidney has returned to its normal position. Glénard's *procède de pousse*, the lifting up of the kidney with the fingers and its palpation with the thumb, is a very simple and easy method of determining displacement of this organ. The tumor made out by these means is usually freely movable, and can be easily replaced. In the case of Dietl's crises a tumor in the lumbar region is frequently made out, which disappears on the cessation of pain. It is an unquestionable fact that too frequent or too severe palpation of the kidneys may harm these organs, and albumin, casts, and blood may be found after such an examination. Einhorn believes that the congestion of the organs due to their displacement may lead to nephritis. We must also remember that a marked change in the size of the palpable tumor is suggestive of an associated hydronephrosis.

In *hepatoptosis* we practically always find a tumor in the right side of the abdomen with its convex surface directed forward. In making the diagnosis we use both palpation and percussion. On percussion the liver dulness, instead of beginning at the fifth rib or fifth interspace in the mamillary line, is at a much lower level, often the costal margin, while the lower limit of dulness is correspondingly depressed. In place of the normal liver note in the hypochondrium and epigastrium, we meet a tympanitic note due to the ascent of coils of intestine. When the patient is made to lie on his back or his right side with the hips elevated, the liver usually falls back into its normal position, and the topographical percussion is normal. For this reason it is important to percuss first with the patient in the erect position. By bimanual palpation we can make out the characteristic tumor in the hypochondriac or lumbar region; the patient must be examined in both the prone and upright position, and an attempt must be made to replace the organ. Because of the rotation of the organ the liver always seems enlarged. Sometimes by inspection a slight bulging of the abdomen on the right side is made out. We must remember that sometimes replacement is impossible

because of adhesions, while in all cases we must differentiate a displacement which is part of a general splachnoptosis from that due to pleurisy with effusion, diaphragmatic abscess, or from enlargement or tumors of the liver. Sometimes we can feel a peculiar tongue-shaped process of the right lobe known as Riedel's lobe.

In diagnosing displacements of the *intestines*, by far the most satisfactory method is the use of the *x*-rays. Usually the cæcum and the transverse colon can be palpated as cords, while inspection will often show us peristaltic waves in the displaced coils. By distention through a rectal tube by air or nitrogen gas we can usually determine satisfactorily the position of the transverse portion of the colon, which is usually in splachnoptosis at or below the umbilicus in close relationship to the greater curvature of the stomach. The pulsation of the abdominal aorta can usually be easily felt. Boas has followed a metallic capsule through the intestinal tract by means of the *x*-rays, while Wordew, Sailer, Pancoast, and Davis have determined the position of the large intestine, and occasionally also a portion of the jejunum, by injecting the bismuth mixture described above through a rectal tube, and subsequently taking an *x*-ray photograph. They especially noted the low position of the hepatic and the high position of the splenic flexure, and the fact that the contents appear to remain longer in the cæcum than elsewhere.

The diagnosis of *splenoptosis* is usually easy, as by palpation the organ with its sharp edge and notch may be easily made out. If, however, the organ is fixed in an abnormal position and deformed by adhesions the diagnosis is much more difficult.

If we make use of the methods described above, the diagnosis of splachnoptosis should be easily made. From a careless examination the condition is frequently regarded as purely a gynæcological one, while in some cases gastroptosis with the cachexia sometimes associated with it has been mistaken for gastric ulcer or carcinoma. Among the conditions which may be mistaken for floating kidney are fæcal tumors in the hepatic flexure or in a loop of the small intestine, a distended gall-bladder, a myoma of the uterus with a long, thin pedicle, or a small ovarian cyst or tumor. Among the conditions for which floating liver has been mistaken may be mentioned renal tumors and hydronephrosis, nephroptosis, and certain rare cases of thickened mesentery or omentum. The spleen has occasionally been mistaken for a uterine myoma, or an ovarian cyst or tumor.

Prognosis.—It must be remembered, in the first place, that in many patients no symptoms are present and no treatment necessary. If they are seen early and are willing and able to carry out the prophylactic measures described, or in those cases in which the symptoms are almost exclusively referable to one displaced organ, real success may attend the treatment, be it medical or surgical; but in the majority of cases seen in adult life, when the organs show considerable displacement, the symptoms are marked and varied and the nervous system considerably involved, although the condition may be markedly relieved in many cases and in some the symptoms made to entirely disappear, yet there will be a large number in whom treatment of any kind will prove very discouraging.

Treatment.—The treatment of splachnoptosis is in the main rather unsatisfactory, particularly so if the symptoms have been present a long time and the displacement of the various viscera has reached a marked degree. To comprehend the proper treatment it is well to remember that

in the majority of cases the condition in all probability depends on a congenital fragility of tissue, while in those so congenitally disposed numerous secondary factors may bring about the displacement of the various viscera.

Prophylaxis.—To prevent marked manifestations of splanchnoptosis it is most important that the tendency should be recognized early and every possible method of prophylaxis carried out. Children with the characteristic body form, or with a slight displacement of any of the abdominal viscera, should be treated with the utmost care; they should be made to lie down during certain hours of the day, especially after meals; great care should be paid to their diet, and if they are extremely thin every effort made to fatten them; the strength of the diaphragm, the intercostals, and the abdominal muscles should be increased by carefully chosen exercises; they should be given breathing exercises, in the hope of enlarging the lower thoracic zone; sometimes they should be made to wear abdominal bandages for a short period of time if their abdominal muscles show great lack of tone; abdominal massage should be given if possible, and everything should be done to put them and to keep them in the best possible physical condition.

Attention should be paid to all persons, whether adults or children, during and after acute or chronic diseases associated with marked loss of weight or diminution of muscular tone, and one should be most careful about allowing such patients to get up from bed and go back to active occupations. Systematic overfeeding associated frequently with massage should be carried out to bring back body weight and muscle tone to normal. Great care should be taken after rapid obesity cures, and it is probable that in this, as well as in a number of the other above conditions, wearing a bandage for a short period of time would be advisable. Too much care cannot be taken during the puerperium; the patient should not be allowed to get up too soon, and when she does she should be provided with a snug abdominal support until the mechanical conditions within the abdominal cavity approximate the normal again. Great care and practically the same precautions should be taken after the removal of large quantities of ascitic fluid, or of large abdominal tumors, for in these conditions, as in pregnancy, there occurs a sudden diminution of intra-abdominal pressure. It is essential that we as physicians should demonstrate to our patients the dangers from the use of tight corsets or tight belts, and if they insist upon their continuance try to persuade them to wear the less unhealthful kind, that is, the straight front corset, where the pressure is exerted upon the lower half of the abdomen. The great majority of patients with splanchnoptosis are deficient in body fat, and even when no symptoms are present we should advise such patients to so regulate their diet and mode of life that an increase in weight will take place. Patients with this tendency should systematically carry out breathing exercises and other exercises to improve the tone of their respiratory and abdominal muscles, while everything should be avoided that tends to lead to pendulous abdomen or to an atonic condition of the abdominal walls.

General Treatment.—The diet in splanchnoptosis depends largely upon the degree to which the stomach and intestines are involved. Glénard advised a careful dietary, depending mainly on meat at first, and gradually adding stale bread and green vegetables, but avoiding acids, wines, cereals, and fats. Ample nutrition to increase the body weight is most important, and this is best obtained by following a simple mixed dietary with little

fluids at meals, while often between meals raw eggs and milk are given; in other cases the patient may be put at first on an absolute milk diet associated with rest. By these methods weight can be added with practically always a marked improvement; we must, however, be extremely careful not to overload the stomach and not to increase the tendency toward dilatation. Often it is advisable to insist that the patient lie down for some time after each meal.

We should advise our patients against wearing tight clothing, and especially against wearing anything that constricts the lower half of the thorax. Great benefit can be derived from carefully selected outdoor exercises and Swedish movements, although these must not be overdone; in fact, it is wise to begin very gently and to gradually increase as the tone of the abdominal muscles improve. Patients should be taught the proper mode of breathing and of standing. Both abdominal and general massage is of real value in this condition, the former either given by a masseuse or by the so-called cannon-ball treatment.

Electricity has been recommended by many especially for the gastric and intestinal symptoms; some have advised intragastric faradization, but this is of doubtful value, because it usually increases the neurasthenic tendencies of the patient.

It has been frequently observed that after rest in bed these patients often show marked improvement, especially in cases of marked gastroptosis with some dilatation. We should remember that rest is one of the most important aids in improving symptoms of this condition, and a strict rest cure often produces splendid results. In some cases during pregnancy the symptoms are markedly relieved by the upward pressure of the enlarged uterus.

Hydrotherapy is often helpful both as regards the gastro-intestinal and the nervous symptoms, and the Scotch douche has been found especially beneficial in this condition. In cases of associated dilatation of the stomach lavage is indicated in association with a careful diet. Taylor believes that alternately using hot and cold water is very beneficial, while McPhedran advises morning and evening massage after the patient has taken one or two glasses of water.

Medicinal Treatment.—Medicine plays but a very small part in the treatment of splachnoptosis. In treating the constipation so frequently met with we should first try massage, electricity, exercise, diet, and hydrotherapy; if these do not prove effectual, enemata may be given, especially those of oil, while if laxatives are necessary, we may use sodium sulphate and aloes, as suggested by Glénard, or Epsom and Rochelle salt, as suggested by Ewald, or any simple laxative. Much benefit is often obtained from the use of alkalies or the drinking of alkaline waters. Intestinal antiseptics are sometimes recommended, but little benefit results from their use. If anæmia is present, iron and arsenic are indicated, although each must be used with care, because they may increase the gastro-intestinal symptoms. As a general tonic nothing is so good as strychnine in increasing doses. As regards water, but little should be taken with the meals, as a rule, but a considerable amount should be taken on waking, in the middle of the morning and afternoon, and at bedtime; the alkaline waters are especially satisfactory. Lincoln, Einhorn, and others advise the use of bromides, while Fuhs gives hydrastis in those cases where the spastic condition of the intestine is marked. In the treatment of the associated anomalies of

secretion we may give alkalis for hyperchlorhydria, hydrochloric acid and bitter stomachics in the more common subacidity and anacidity. Ruggi gives kidney extract in nephroptosis, believing it is due to perversion of the renal secretions.

Treatment by Mechanical Supports.—In the hands of most clinicians the use of various mechanical supports in association with the measures already described has proved of great value. The object of the support is, of course, to lessen the volume of the lower half of the abdomen and to increase intra-abdominal pressure; to get satisfactory results it is essential that the corset or belt should be applied with the patient in the inclined dorsal position, so that the various viscera are in their position of least descensus. The support is dangerous if it is not well made or applied properly, because if the pressure is above any of the displaced viscera it increases the descensus, and unless the patient carries out systematic abdominal exercises the use of the bandage is likely to decrease the tone of the abdominal muscles. Morris especially insists upon the increased pain in nephroptosis on wearing a bandage. As to the value of the bandage, some authors hold that it is absolutely curative, others that it is palliative at best; as a rule, when its use is associated with the proper dietetic, hygienic, and medicinal measures, a disappearance of many of the symptoms results in the majority of cases. Aaron reports 600 cases of nephroptosis, 90 per cent. of which were relieved by this method, and 215 of which, according to him, had an absolute recovery. Francine believes that uncomplicated cases of gastropptosis practically all recover under the use of the bandage. It is difficult to see, however, how, anatomically speaking, a real recovery is possible by such means, although there may be a complete disappearance of symptoms. The bandage is especially useful when the ptosis is due to weakness of the abdominal wall.

Many different kinds of bandages have been recommended: Glénard advises an elastic bandage with straps. Rose believes that the best bandage is one made of adhesive plaster, a view agreed to by Kemp, Meade, Clemm, and Rosewater. Gallant advises a corset made to fit the patient and laced from below upward with the patient in the inclined dorsal position. Ewald and Kuttner use besides the bandage a pad in kidney displacements, while Riegel usually uses a pad or bolster in gastropptosis. Among abdominal supporters may be mentioned those of Teufel and Longstreth, the belt of the latter being strongly recommended by Taylor. The object of all these supports is, of course, the same; the pressure is applied in the lower portion of the abdomen, and acts from below backward and upward, while the intestines are held up and act as a pad against the other abdominal viscera.

Operative Treatment.—It has always been a moot question whether splanchnoptosis is a medical or a surgical condition. It seems that in the vast majority of cases it is strictly medical, and only surgical when the symptoms are so definitely referable to one or two organs that their suspension will offer a good chance of the disappearance of the symptoms; in *every* case hygienic, dietetic, and mechanical measures should be faithfully tried first. Among the indications for surgical interference may be mentioned those cases where practically all the symptoms are referable to the displacement of one organ alone, which is sometimes seen in gastropptosis with its associated discomfort after eating, anorexia and cachexia, and gastropptosis with marked dilatation. In the case of the kidney, Dietl's crises, intermittent hydronephrosis, symptoms suggestive of renal or hepatic colic,

more or less constant pain, symptoms due to pressure of the displaced kidney upon other organs, or upon the sympathetic or genitocrural nerves, are all indications for operative treatment; and also, as Kelly has shown, when the attacks of pain may be reproduced by injecting salt solution into the renal pelvis through a ureteral catheter. Compression of the ovarian vein, with subsequent pelvic disease, as insisted on by Goelet, is also a sufficient reason. In the case of the liver, pain of a tearing, twisting character if frequent, attacks like hepatic colic, and symptoms due to pressure on other organs, in the case of the spleen, torsion of the pedicle with swelling and gangrene, in the case of the intestines, constipation which does not yield satisfactorily to any medicinal or mechanical treatment, are all indications for surgical measures. It seems also justifiable to try operative measures in those cases with marked symptoms where all other methods have been entirely devoid of success.

In operating upon the *stomach* various methods have been devised. Duret, in 1896, stitched the stomach to the abdominal wall; Lambotte attached by suture the splenic and hepatic flexures to the abdominal wall, thus lifting up the stomach; Webster resected the abdominal wall, bringing together the recti, which had undergone diastasis; Walker performed gastrojejunostomy; Eve operated by placing sutures through the lesser curvature, the lesser omentum, and the liver substance; Coffey by suspending the stomach in a hammock made by the great omentum; Blecher and Bier by shortening and folding the gastrohepatic ligament. Stitching of the stomach to the diaphragm has been done, and Beyea has passed sutures through the gastrohepatic and gastrophrenic ligaments, the stomach by this method not being fixed, but elevated into its normal position without affecting its mobility; while in cases of gastropptosis with marked dilatation, gastro-enterostomy or perhaps in some cases pyloroplasty, may be performed. It must not be forgotten that in some of these cases fixation is followed by dilatation, persistent vomiting, and even by marked thinning of the stomach wall. Rovsing reports 54 gastropexies, with good results in each, while Beyea reports 11 cures in 11 cases by his method.

As regards fixation of the *kidney*, many methods have been described, such as suture through the fatty capsule alone, through the fibrous capsule alone, through the kidney substance, splitting and dissecting the capsule and stitching it to the lumbar muscles, and packing with gauze, which is kept in until firm adhesions are formed. Some of the older surgeons performed nephrectomy, and of 59 cases so treated 19 died. Hahn did the first nephropexy in 1881. Most operators now advocate suture of the parenchyma in addition to a free dissection of the fibrous and fatty capsule, while Kelly insists upon the Brödel stitch. Wehrmann and Harris believe that the tendency has been to stitch the kidney too high, as the liver may again press upon the upper pole and bring about a recurrence of the condition. Relapses after nephropexy are due either to this or to insufficient fibrous union between the kidney and the lumbar muscles, which yields under gravity or the pressure of corsets, while in some cases the kidney is fixed in the wrong position and the function of ureter and bloodvessels markedly interfered with; in women a satisfactory nephropexy is peculiarly difficult because of the greater obliquity of the lower ribs. Recently considerable attention has been paid to the association between right nephropptosis and chronic appendicitis, and some operators after stitching the kidney into positions remove the appendix if the symptoms have been at all suggestive.

The mortality in nephropexy is practically nil; thus, Keen reports 4 deaths in 134 cases; Edebohls, none in 193; Morris, none in 198; Goelet, none in 126; Sturndorff, none in 83; Rovsing, none in 100; while, as regards the success of the operation, Goelet reports 126 cures in 126 cases, and Hahn, in his series of 900 cases collected from his own service and from the literature, finds cures in from 2 to 60 per cent. of the cases according to the series. Burnam finds that the result of the anatomical fixing method is practically perfect, there being no failures if the operation is properly done. As to the results, he found that 90 per cent. were cured of pain, about the same number showed a relief of stomach symptoms, while 50 per cent. were improved nervously; the results where both kidneys were movable were practically the same as in the case of unilateral disease if both were suspended. Of course, it must be remembered that in this, as in many other series of floating kidney operations, in the majority of cases operation was advised because of local symptoms. Tuffier has found that the result of fixation of displaced kidneys when hydronephrosis is present is not so good as in cases where it is absent, for while the operation relieves the pain, the distention if present for long has affected the integrity of the organ, and it never regains its normal tone. Treves believes from a study of 300 cases that suturing of the kidney should be a rare operation, as he believes as good, if not better, results are obtained by the use of mechanical supports, a view which is also held by Israel. McWilliams found little benefit in the majority of 61 cases operated on, while Balch and Torbert found that only 28 out of 86 cases operated upon at the Massachusetts General Hospital were relieved.

As regards fixation of the *liver*, hepatopexy, various operations have been devised and performed. Billroth and others have sutured the liver to the abdominal wall, Langenbech has selected the lower costal cartilages as the points of fixation, some have produced adhesions between the convexity of the liver and the diaphragm by irritation of their surfaces, while others have sutured the fundus of the gall-bladder to the parietal peritoneum. Mayer reports 6 cases of hepatopexy done by Depage, where a strip of peritoneum 5 cm. wide was detached and sutured to the posterior aspect of the liver margin; Franke, in addition, passes a few sutures through the liver substance. Rovsing has reported 18 hepatopexies with good results. In most cases movable *spleen* requires no special treatment, but where there is torsion of the pedicle and symptoms of stasis, or where there are symptoms of pressure upon other organs, splenectomy has been done.

In displacement of the *intestines* considerable surgical work has been done recently. In the case of the small intestines, filled with liquid fæces, ptosis produces comparatively slight symptoms, but the presence of solid fæces in the large intestines means that descensus is frequently associated with persistent and intractable constipation, this being especially so in ptosis and kinking of the sigmoid. To counteract this various operations have been performed, such as resection and anastomosis of the colon, suspension of the sigmoid flexure (sigmoidopexy) and even resection of the sigmoid; the results in the main have been extremely satisfactory.

CHAPTER IX.

DISEASES OF THE PANCREAS.

By EUGENE L. OPIE, M.D.

GENERAL PATHOLOGY AND SYMPTOMS.

ALTHOUGH the pancreas is essential to the normal digestion of proteids, carbohydrates, and fats, and controls the assimilation of carbohydrates, in only a small proportion of cases does disturbance of these functions indicate with certainty disease of the organ. Inaccessibility to palpation prevents the recognition of alterations in size, while contact with a variety of important structures diminishes the value of subjective symptoms as a means of localization. Disease of the pancreas is rarely uncomplicated, and, being in many instances the result of disease of adjacent structures, its symptoms are obscured by those which accompany the primary disease. On the one hand, lesions of the stomach, duodenum, liver, and bile passages are frequent causes of pancreatic disease, while, on the other hand, lesions in the gland may cause secondary changes in all of these organs.

Evidence of disturbed function is obtainable only when the gland is the seat of advanced disease. Complete extirpation of the gland in animals is followed both by glycosuria and by changes in the digestion of proteids and fats; partial extirpation may be unaccompanied by glycosuria or by disturbances of digestion, since a small part of the gland has been found to fulfil both the internal or metabolic and the external or secretory function of the gland. The impairment of function resulting from slight injury may be unrecognizable under ordinary conditions, and demonstrable only by special means which tax the capabilities of the organ. Moreover, complete occlusion of the duct of Wirsung, which is usually the larger duct of the gland, does not necessarily prevent access of the secretion to the intestine, since the duct of Santorini, anastomosing with it, may be large enough to act as an outlet for the entire secretion.

Symptoms Due to Impairment of Pancreatic Secretions.—Symptoms referable to loss or impairment of function concern (1) the external secretion of the gland, indicated by disturbance of digestion or by the effects of retained secretion, and (2) the internal function of the organ, causing disturbance of carbohydrate metabolism. Since the pancreatic juice contains ferments which are concerned in the digestion of proteids, fats, and carbohydrates, impaired absorption of these constituents of the food may follow destruction of the secreting parenchyma or such occlusion of the ducts that pancreatic juice no longer enters the duodenum. Pancreatic juice, however, is not the only secretion concerned in the digestion of any one of the constituents of the food which have been mentioned.

Steatorrhea.—Kuntzmann (1820) first observed fatty stools with disease of the pancreas. His patient, who suffered with jaundice, had on several

occasions passed fat in large quantities; at autopsy the pancreas was found to be indurated and the duct of Wirsung obliterated. Bright (1833) subsequently published 7 cases of pancreatic disease, in 3 of which this symptom was present. By injecting oil into the pancreatic duct of dogs, Claude Bernard succeeded in causing atrophy of the pancreas, and observed that the fæces contained both undigested meat and fat, at times in such quantity that when cold it formed a layer over the fæcal mass. Although steatorrhœa has been regarded by Friedreich and other writers as a symptom of much diagnostic value, they have recognized the fact that it accompanies only a small proportion of even grave pancreatic disorders. Finding jaundice associated with cases of pancreatic disease, in which steatorrhœa indicated impaired absorption of fat, Fr. Müller has attributed the symptom to obstruction of the common bile duct. A review of recorded instances of steatorrhœa with pancreatic disease, made by Fitz¹ in 1903, does not confirm this view, but establishes the diagnostic value of the symptom in the relatively small number of cases in which it occurs. He was able to collect from the literature 29 cases in which autopsy, laparotomy, or the passage of a pancreatic calculus gave evidence of disease of the gland, and in only 12 of these cases was jaundice found. In the 17 cases in which jaundice was absent the lesion of the pancreas was in 7 cases tumor, usually cancer; in 6 cases calculi within the ducts; in 2 cases cysts with atrophy; in 2 instances the lesion was designated fatty degeneration.

In the recorded cases the character of the fæces has varied considerably; in some instances superficial examination demonstrates the presence of free fat, which is described as oily or like butter, while in other cases the fæces are described as gray, silvery gray, or asbestos-like. The fat, which is liquid when passed, may solidify on cooling and form a layer covering the fæcal mass. The presence of fat recognizable by the eye constitutes a true steatorrhœa.

In the uniform metallic gray or asbestos-like stools sometimes seen, the abnormal fatty contents are demonstrated with greater certainty by chemical methods which may show the amount of neutral fat, fatty acids, and soaps. Such fatty stools may occur in normal individuals who have ingested fat in quantity so great that the normal limit of absorption has been exceeded. Ability to digest and absorb fat is diminished, according to Nothnagel, when bile fails to enter the intestine, when absorption is hindered by certain diseases of the intestine or its lymphatic apparatus, namely, amyloid disease, extensive atrophy of the mucous membrane, caseation of the mesenteric lymphatic glands, and tuberculous peritonitis, or when very active peristalsis prevents the normal action of the digestive juices.

Although Müller found in 2 cases of pancreatic disease no increase in the percentage of fæcal fat, chemical analysis demonstrated a diminution of split fat. In health, according to his observations, the unabsorbed fæcal fats consist of approximately from 20 to 30 per cent. of neutral fat and from 70 to 80 per cent. of split fat, which is partly fatty acids and partly soaps. In his two cases the split fat was diminished to 22.4 and 47.7 per cent. respectively. In some cases of pancreatic disease access of pancreatic juice in considerable amounts to the intestine, even though the duct is partially occluded or the parenchyma partially destroyed, may explain the

¹ *Transactions of the Congress of American Physicians and Surgeons*, 1903, vi, 36.

absence of diminished splitting of fat. Katz¹ has maintained that diminution of fatty acids and soaps below 70 per cent. of the total amount of faecal fat indicates diminished action of the pancreatic juice, and only in nursing infants and in patients with profuse diarrhoea is the diagnostic significance of this condition lost. Fitz has collected from the literature 11 cases of pancreatic disease, confirmed by autopsy, in which analyses of the faecal fat had been made during life; in 7 instances jaundice was absent. The total amount of fat determined in 4 instances was slightly increased, but in no case apparently was there macroscopic steatorrhoea. The percentage of neutral fat in 7 instances of uncomplicated disease of the pancreas was normal or less than normal in only 1 case, and although the results of such examination have not been constant, a well-marked diminution in the proportion of split fat has been usually present in such cases.

Macroscopic or chemical evidence of diminished digestion and absorption of fat, obtained by examination of the faeces, indicates the presence of pancreatic disease when other disturbing influences, such as jaundice or intestinal diseases preventing absorption or hastening peristalsis, can be excluded. In the absence of macroscopic fat in the faeces, demonstration of diminished splitting of fat by chemical examination may suggest the presence of pancreatic disease. With the data at present available little significance can be attached to the proportion of fatty acids to soaps, although Zoja² found diminution of the latter with pancreatic disease.

The capacity of different individuals to digest fat varies within such wide limits that little information concerning impaired power of digestion can be obtained by observing the effect upon the faeces of the administration of a fixed amount of fat. Hartsen found no increase of faecal fat after administration of from eight to ten teaspoonfuls of cod-liver oil to two diabetics, in each of whom autopsy showed advanced atrophy of the pancreas.

By administering fresh pancreas to animals from which the pancreas has been removed the consequent impairment of fat absorption has been diminished. Abelman found that the pancreas of the pig fed to a dog, the pancreas of which had been extirpated, caused a decrease in the amount of undigested fat and of proteid as well in the faeces. Similar observations have been made by other experimenters, and are confirmed by a few observations made upon patients suffering with pancreatic disease. Fles, quoted by Friedreich, records the case of a man suffering with diabetes mellitus who passed undigested meat and fat in such quantities with the faeces that it could be separated from the faecal mass by ounces. A part of the milky fluid obtained by rubbing in a mortar half of the fresh pancreas of a calf with six ounces of water and straining the mixture was administered after each meal in such quantity that one pancreas was consumed daily. At the end of two days all fat had disappeared from the faeces and the number of undigested fibers of striped muscle was greatly diminished. Whenever the administration of the infusion was discontinued, fat and muscle fibers reappeared in the faeces. The condition of the patient improved for a time, but diabetes mellitus persisted, and death occurred as the result of phthisis. Advanced sclerosis of the pancreas was found at autopsy. Oser observed improved digestion of fats following the administration of one gram of

¹ *Wiener med. Woch.*, 1899, xlix, 153.

² Quoted by Oser, *Die Deutsche Klinik*, 1901, v, 151.

pancreatin (Merck) taken daily in divided doses; there was a tumor mass in the epigastrium, jaundice, and impaired digestion of fat and proteid indicated by their presence in the fæces, but the diagnosis of carcinoma was not confirmed by anatomical examination.

Little is known concerning the effect of continued steatorrhœa upon nutrition and health. Walker has described the case of a man who for twenty years passed large colorless stools of putrid odor, containing free oily or solid fat. There was no jaundice. During this time he was in good health and engaged in the practice of medicine; there was no marked emaciation. He died at the age of ninety years. The pancreas was apparently replaced almost wholly by fat, and its duct was occluded by an irregular calculus situated about an inch from the duodenum.

Azotorrhœa.—The presence of undigested proteid material in the fæces, so-called azotorrhœa, has been less frequently observed in association with pancreatic disease than disturbed digestion of fat. After causing almost complete destruction of the pancreas in dogs, Claude Bernard found undigested muscle fibers and even large pieces of meat in the fæces. Abellmann found that less than 2 per cent. of the nitrogen of the food reappears in the fæces of normal dogs; when the pancreas is completely removed, 56 per cent. of ingested nitrogen was lost with the fæces, whereas after partial removal 46 per cent. was lost. When the pancreas of the pig was administered, loss of nitrogenous material was diminished to approximately 24 per cent. Other observers have obtained similar results.

In cases of diabetes due perhaps to lesions of the pancreas, Hirschfeld recovered in the fæces about 32 per cent. of the nitrogen ingested with the food (the percentage normally present being 5 or 6), but the presence or absence of pancreatic disease was not determined by autopsy. In a case described by Wientraud autopsy demonstrated advanced chronic interstitial pancreatitis; in the fæces during life he recovered 45.2 per cent. of the ingested proteid and only 22.2 per cent. of the fat taken as food. He suggests that pancreatic disease causes as great disturbance of the digestion of proteids as of fats, perhaps an even greater disturbance, and is therefore distinguishable from intestinal disturbances such as amyloid disease, caseation of the mesenteric lymph glands, etc., which hinder absorption of fat alone. With closure of the duct of Wirsung by carcinoma, Zoja recovered from the fæces nitrogenous material equal in amount to 70 per cent. of that taken in the food.

The presence of undigested meat in the fæces may give indication of disturbed proteid digestion. Fles found well-preserved muscle fibers in the fæces of the patient previously mentioned, and administration of an extract made from the pancreas of a calf caused their disappearance. Fitz was able to collect from the literature of the subject only 8 cases in which the presence of undigested muscle fibers in the fæces had been associated with clearly demonstrated pancreatic disease; in 5 of these cases steatorrhœa was present. The condition, as he points out, probably occurs only when there is extreme diminution of the pancreatic juice, and is significant only when gastric digestion is normal, when the diet contains no excess of meat, and when there is no diarrhœa.

The *persistence of nuclei in remnants of muscle fibers passed with the fæces* has been proposed by Adolph Schmidt as a test of the efficiency of pancreatic digestion. He maintains that the nuclear material of meat can be digested only by the pancreatic juice, and the presence of nuclei in muscle

fibers expelled with the feces indicates defective pancreatic secretion. Slightly fibrous beef cut into small cubes one-half centimeter square is hardened in absolute alcohol, and after enclosure in silk gauze is preserved in alcohol. The enveloped cubes, after washing for three hours in water, are administered to the patient in wafers together with the special diet recommended by Schmidt. If in case of pancreatic disease muscle remains in the sacs recovered from the feces, it is dehydrated in alcohol and examined after embedding in sections stained by nuclear dyes; or the specimen is simply washed in water and, after teasing, treated with acetic acid or methylene blue.

Beef muscle so prepared failed to lose its nuclei when passed through the intestinal canal of three dogs from which the pancreas had been removed. Steele¹ found the test little more than an index of proteolytic digestion in the intestine, giving only vague information concerning the pancreatic secretion.

A method for testing the efficiency of proteid digestion has been devised by Sahli. Gelatin capsules sufficiently hardened in formol have been found to resist gastric digestion during twelve hours or more, but are rapidly dissolved by pancreatic juice. Such capsules are filled with iodoform, and at various intervals after their administration the urine or saliva is tested for iodine. In normal individuals the reaction appears in from four to eight hours, and, according to Sahli, delayed reaction indicates an impairment of pancreatic digestion, provided the mobility of the stomach is normal. Impaired intestinal digestion may likewise delay the reaction, and with diarrhoea undigested capsules may appear in the feces. Fromme² doubts the diagnostic value of the reaction, but in neither of his cases was the relation of the lesion to the patency of the pancreatic duct clearly established.

It has been believed that the formation of certain substances which occur as the result of putrefaction of proteids in the intestine is favored by presence of pancreatic juice. Unchanged proteid, it is maintained, is little altered by intestinal bacteria, while products of pancreatic digestion are readily decomposed. Hence a diminished quantity of such products of decomposition, notably *indican* and *ethereal sulphates*, in the urine may give indirect evidence of impaired pancreatic secretion. Gerhardt found indican absent in the urine of a patient suffering with intestinal obstruction, and attributed its absence to acute hemorrhagic pancreatitis and occlusion of the pancreatic duct which were found at autopsy. Diminution of indican in the urine has not been found to be a constant sequence either of experimental extirpation of the gland or of pancreatic disease. Herter and Northrup found an increased amount of indican in the urine of a patient suffering with carcinoma of the pancreas.

Edsall³ thinks that diminution of ethereal sulphates in the urine should suggest pancreatic disease when conditions are present which usually cause their increase. He recognizes, however, that ethereal sulphates may be diminished with a variety of diseases which do not affect the pancreas. In favor of his opinion he cites a case of carcinoma of the pancreas, demonstrated by autopsy, and the cases of Le Nobel and of Taylor, in which the same lesion was probably present.

¹ *University of Pennsylvania Medical Bulletin*, 1906, xix.

² *Münchener med. Woch.*, 1901, xlviii, 591.

³ *American Journal of the Medical Sciences*, 1901, cxxi, 401

By applying the phenylhydrazine test for sugar to urine previously boiled with strong hydrochloric acid and treated with basic lead acetate, in order to remove glycuronic acid, Canmidge¹ has obtained sheaves of yellow crystals soluble in dilute sulphuric acid which he believes indicate the presence of pancreatic disease; their nature is undetermined. The urine of 29 patients gave a positive reaction, and chronic pancreatitis was diagnosed at operation. In four instances of what was believed to be carcinoma of the pancreas the reaction was absent, while in one case it was present; 67 times no crystals were found, and there was no evidence of pancreatic disease. Taylor² has obtained the reaction in cases in which there has been no evidence of pancreatic disease; Eichler³ has obtained the reaction in the urine of dogs with experimental pancreatic disease.

Impairment of the Metabolic Function of the Pancreas.—Glycosuria and diabetes mellitus are so frequently associated with pancreatic disease that it is necessary to consider them among its symptoms and to define the peculiarity of those lesions which cause disturbance of carbohydrate metabolism. In order to understand their significance for diagnosis of disease of the organ and their relation to disturbance of its secreting function, it is essential to discuss briefly their relation to the different histological structures which make up the gland. The relation of the islands of Langerhans to carbohydrate metabolism has been described in detail in the chapter on Diabetes Mellitus.

Study of the histological anatomy of pancreatic disease has furnished, for the first time, data by which it is possible to define the anatomical basis of diabetes mellitus. In 1904 Sauerbeck⁴ was able to collect from the literature of the subject 176 cases of diabetes mellitus studied by observers who have given attention to the islands of Langerhans; it has been possible to collect 112 additional cases which have since become available. Analysis of these cases gives more accurate statistics concerning the relation of various pancreatic lesions to diabetes than has been obtainable before. Nevertheless, figures which indicate the frequency of various lesions are only approximate, for many writers have not described their cases in detail, and in some instances different writers have described the same change under different names. For example, the same lesion has been designated atrophy and chronic pancreatitis, and rarely has a distinction been made between atrophy causing diminution in the size of the gland and congenital smallness of the gland. The lesion designated lipomatosis introduces further error, for in some cases it indicates a moderate increase of the interstitial fat, and is a trivial abnormality which is not responsible for the existing glycosuria, whereas in other cases increase of fat is the result of interstitial inflammation, and is accompanied by sclerosis or hyaline degeneration of the islands of Langerhans; from the available records, it is often impossible to classify such cases. The findings are as follows:

¹ *British Medical Journal*, 1906, i, 1150.

² *Lancet*, 1906, i, 1818.

³ *Berliner klin. Woch.*, 1907, xlv.

⁴ Lubarsch and Ostertag, *Ergebnisse der allgem. Path.*, 1904, viii, 588.

Interacinar pancreatitis	123
Atrophy	65
Lipomatosis	18
Interlobular pancreatitis with no occlusion of ducts	13
Lithiasis	9
Cyst	1
Carcinoma	5
Focal necrosis	2
Secreting parenchyma normal or approximately normal:	
Hyaline degeneration of islands of Langerhans	6
Sclerosis " "	4
Diminution " "	5
Hypertrophy " "	3
Normal	34

288

Hypertrophy of the islands of Langerhans which has been observed with diabetes mellitus is of two kinds: (1) Simple hypertrophy, which accompanies destructive lesions of these structures (Schmidt, Pearce¹), and (2) adenoma-like hypertrophy, which may occur in young individuals without other lesions of the islands of Langerhans (Herxheimer, Reitmann, MacCallum²). Hypertrophy doubtless occurs because existing islands of Langerhans are insufficient to meet functional demand.

It is noteworthy that in a considerable number of cases of diabetes no lesion of the pancreas is demonstrable by careful microscopic examination. Experimental and pathological data establish with certainty that diabetes mellitus may be the result of pancreatic disease, and save in a comparatively small percentage of cases this origin is demonstrable. The fact that the islands of Langerhans and secreting parenchyma in a group of cases are normal does not oppose the theory that lesions of the former are responsible when the pancreas is the source of the disease, for it equally opposes any theory of pancreatic diabetes. It is not improbable that complex carbohydrate metabolism may be disturbed by factors which do not concern the pancreas. Diabetes in such cases is not, however, distinguishable during life from so-called pancreatic diabetes. It is further noteworthy that the proportion of cases in which diabetes is referable to the gland has been materially increased by recent histological studies; the statistics of Windle (1883) contain 46.8 per cent. of cases with normal pancreas.

The foregoing considerations make it possible to define with greater accuracy the conditions under which glycosuria and other symptoms of diabetes occur as symptoms of pancreatic disease, and to determine the relation which diabetes holds to steatorrhœa, azotorrhœa, and other evidence of an insufficient supply of pancreatic juice. Since sclerosis of the islands of Langerhans or hyaline degeneration of these structures may occur in the presence of little or no change affecting the secreting acini, diabetes may occur without digestive symptoms of pancreatic insufficiency, and indeed, the greater number of cases of pancreatic diabetes belong to this group. Destructive lesions, such as abscess, acute hemorrhagic pancreatitis and diffuse carcinoma, invading the gland, destroy equally islands of Langerhans and secreting acini, and in the majority of instances affect a previously healthy gland. The

¹ *Albany Medical Annals*, 1904, xxv, 329.

² *American Journal of the Medical Sciences*, 1907, cxxxiii, 432.

resulting condition resembles that which follows partial extirpation, and is accompanied by diabetes only in the comparatively rare cases in which almost the whole gland is destroyed.

Occlusion of the pancreatic ducts does not cause diabetes, and the resulting chronic interlobular pancreatitis is accompanied by the disease only when it has reached the advanced stage, in which the parenchyma is largely replaced by dense fibrous tissue. When the pancreatic ducts have been occluded by calculi or by new-growth, steatorrhœa and perhaps other symptoms of deficiency of pancreatic juice may occur without diabetes; of the 29 cases of steatorrhœa collected by Fitz, in 11 diabetes was present. It is not improbable that the application of chemical methods to the examination of feces would demonstrate insufficiency of pancreatic digestion in a much larger number of instances of duct obstruction at a time when diabetes has not appeared.

Since chronic interlobular inflammation which results from occlusion of the pancreatic duct produces diabetes gradually, cases occur in which the associated diabetes is of a mild type and disappears when a diet poor in carbohydrates is administered. Such cases have been described with calculus by Lichtheim and Opie, and with cyst by Lazarus.

Histological studies have, moreover, explained the relationship of a variety of diseases, such as arteriosclerosis and cirrhosis of the liver, to diabetes mellitus. Grube found arteriosclerosis 66 times among 117 cases of diabetes. Chronic interstitial pancreatitis accompanying sclerosis of the pancreatic vessels has been described; this change which affects the interacinar tissue of the gland implicates the islands of Langerhans. G. Hoppe-Seyler,¹ who has studied 18 such cases, found a close relation between the severity of the glycosuria and the degree of change affecting the islands of Langerhans. The frequency with which diabetes is caused by disease of the arteries is shown by 112 cases of diabetes which have been recently recorded; in association with general arteriosclerosis there was, in 23 cases, sclerosis of the pancreatic arteries and interacinar pancreatitis; in 26 additional cases there was either general or local arterial sclerosis, in 10 instances with interacinar pancreatitis.

The frequency with which diabetes mellitus accompanies cirrhosis of the liver has been explained by the presence of chronic pancreatitis. Numerous studies have shown that conditions which cause both atrophic and hypertrophic cirrhosis of the liver act upon the pancreas and cause chronic inflammation. The literature of the subject contains numerous instances of this association, which is well exemplified by the disease known as hæmochromatosis; pigmentary cirrhosis of the liver is accompanied by chronic pancreatitis, which invades the islands of Langerhans and causes diabetes (*diabète bronzé*).

The association of diabetes mellitus and acromegaly is doubtless explained by changes in the pancreas. Chronic inflammation has been observed repeatedly, but the condition of the islands of Langerhans has been noted only by Norris,² who found hyaline degeneration, although the parenchyma was normal.

That *alimentary glycosuria* demonstrable by the administration of a

¹ *Deutsches Arch. f. klin. Med.*, 1904, lxxxi, 119.

² *Proceedings of the New York Pathological Society*, 1907, vii, 19.

fixed quantity of sugar may accompany a variety of pancreatic lesions is shown by the investigation of Wille. From 70 to 100 gm. of grape sugar dissolved in half a liter of coffee were administered in the morning before other food had been taken to 800 patients suffering with a great variety of diseases. Subsequently 77 of them came to autopsy. Temporary glycosuria had been produced in 15 of these, and grave lesions of the pancreas were found in 10, namely, chronic interstitial inflammation, or carcinoma of the gland, either primary or secondary to tumors in the stomach, liver, or gall-bladder. It is well known that alimentary glycosuria may occur in the absence of pancreatic lesion with a variety of diseases, especially with hysteria and other neuroses, chronic alcoholism, and exophthalmic goitre. Its occurrence, however, may give information when other conditions suggest the presence of pancreatic disease of moderate intensity. Hoppe-Seyler, who has shown that arteriosclerosis causes chronic pancreatitis, which in turn causes diabetes when lesions of the islands of Langerhans are sufficiently advanced, has tested the ability of individuals with arteriosclerosis to assimilate 100 gm. of glucose. Alimentary glycosuria was present in 42 per cent. of 62 patients with arteriosclerosis, and was more frequently observed when arteriosclerosis was associated with acute alcoholism.

FAT NECROSIS.

The lesion known as fat necrosis, characterized by death of fat cells in sharply circumscribed areas, is frequently associated with disease of the pancreas. By some writers (Balser) it has been regarded as an often fatal disease accompanied by certain somewhat indefinitely defined symptoms. The lesion when widely disseminated is, in the greater number of instances, the result of acute hemorrhagic pancreatitis. Its recognition is of great importance to the surgeon, since the presence of the characteristic opaque white foci studding the translucent abdominal fat will explain the nature of otherwise obscure conditions for the relief of which laparotomy has been performed.

The monograph of Friedreich (1875) contains no reference to this condition, but from the older literature Fitz quotes the case of Hooper (1861) who apparently attempted to describe the lesion. The first adequate description is that of Balser, who records the appearances found in two individuals dead as the result of a hitherto undescribed disease. In one case necrosis of retroperitoneal fat was associated with sequestration of the pancreas, while in the second case hemorrhage about the pancreas was believed to be the result of the same lesion. Balser, who suggested that very fat individuals are specially susceptible, attributed it to overgrowth of fat cells destroying tissue which they surround. Chiari subsequently added facts concerning the pathological anatomy of the lesion, and observed its presence in 5 of 6 cases of pancreatic disease, in 3 of which the pancreas had undergone sequestration. Fitz clearly recognized the frequent association of fat necrosis with hemorrhagic and gangrenous pancreatitis, and found it much less frequently associated with suppurative inflammation.

Etiology.—The inconstant results of bacteriological examinations do not support the opinion of a few observers (Ponfick), who have maintained that the lesion is caused by bacteria. Its relation to bacteria is doubtless

that suggested by Welch, who obtained the colon bacillus from foci of fat necrosis in association with hemorrhagic pancreatitis; diphtheritic and ulcerative colitis apparently had facilitated the entrance of *Bacillus coli*, which was found not only in the patches of necrotic fat, but in the mesenteric lymph glands, liver, bile, lungs, spleen, and kidneys.

Experimental Fat Necrosis.—Studies of R. Langerhans first brought the lesion into relation with the peculiar physiology of the pancreas. He was able to demonstrate by chemical and histological studies that the fat of the necrotic cell is split into fatty acids, which are deposited as needle-like crystals, and glycerin, which is absorbed. The occurrence of the lesion in association with disease of the pancreas, its greatest intensity in the neighborhood of the gland, and the changes demonstrable within the fat cells have suggested the probability that the pancreatic ferments, particularly the fat-splitting ferment, have an important part in its production. By injecting an emulsion of pancreatic tissue into animals, Langerhans succeeded in producing a small focus of fat necrosis, but more successful results were obtained by Hildebrand and Dittmer, who subjected the pancreas to a variety of injuries; by placing a ligature about the gland, foci of necrosis were obtained in the fat about the part of the gland distal to the ligature, while if the veins of the ligated parts were tied, thus preventing, as they suggested, possible absorption of the obstructed secretion, foci of fat necrosis were even more abundant. If the gland were simply cut across, thus allowing the escape of the pancreatic secretion, foci of necrosis were found in the immediate neighborhood of the gland. If penetration of pancreatic secretion into fat tissue is facilitated by transplanting the duodenal part of the pancreas containing the severed ends of the ducts into the subcutaneous fat of the abdominal wall in cats, extensive subcutaneous necrosis of fat is thus produced (Opie).

In these experiments conditions have been present which afford an opportunity for the escape of pancreatic secretion into the tissue surrounding the gland. A ligature about the gland obstructs the outflow of the pancreatic secretion, which may be forced into the tissues near the gland in the same way that bile finds its way into the interstitial tissue of the liver when the common bile duct is occluded; injury to the parenchyma by cutting or tearing the tissue of the organ allows the secretion to escape from the normal channels. Flexner has demonstrated the presence in the necrotic foci of a ferment capable of splitting neutral butter fat and setting free acids which are recognizable by suitable tests. That the lesion is caused by a ferment is further shown by the experiments of Wells;¹ watery extracts of the pancreas of the hog constantly produce fat necrosis in cats and dogs, but fail to do so when boiled or after heating to a temperature above 71° C. Whether the lesion is wholly due to the action of the fat-splitting ferment or is the result of its action on tissue previously injured by some other constituent of the pancreatic juice, perhaps by the proteolytic ferment, is not yet definitely determined. With preparations of trypsin having no lipolytic action Wells failed to produce the lesion, although he was successful with commercial pancreatin and with other preparations exhibiting lipolytic but no proteolytic activity.

Incidence of Fat Necrosis.—In human cases extensive fat necrosis is more frequently associated with hemorrhagic and gangrenous pancreatitis

¹ *Journal of Medical Research*, 1903, ix, 70.

than with other lesions of the gland, and constantly accompanies acute hemorrhagic pancreatitis produced experimentally by the introduction of a great variety of irritant substances into the gland. The well-known fact that bile forms a favorable medium for the action of the pancreatic ferments, and particularly of the fat-splitting ferment, the activity of which may be increased by it twofold or threefold, probably explains in part the extent of the lesion accompanying many cases of hemorrhagic pancreatitis which may be caused by the penetration of bile into the pancreas. Fat necrosis has been found in association with obstruction of the duct of Wirsung caused by pancreatic calculi, by biliary calculi, or by carcinoma invading the head of the pancreas, but in such cases foci of necrosis are not abundantly distributed, nor does the lesion usually follow obstruction of the pancreatic duct. In a small proportion of cases chronic interstitial pancreatitis is accompanied by fat necrosis, which is probably caused by constriction of small branches of the pancreatic duct.

In several cases of extensive fat necrosis no noteworthy lesion of the pancreas has been demonstrable. In such a case described by Fraenkel the lesion was explained by a gallstone lodged in the diverticulum of Vater, while in a somewhat similar case of Flexner's, gallstones were present in the common bile duct near its termination. A unique case described by Wulff¹ is not in accord with other observations and with present knowledge of the lesion is unexplainable, unless an incomplete autopsy failed to disclose an aberrant pancreas, biliary calculi, or some cause of duct obstruction.

Pathological Anatomy.—A feature of the lesion is its occurrence in sharply defined foci. On opening the abdomen the omentum and perhaps (fat) in other situations is found to be studded with rounded or oval areas, which vary in diameter from less than one to five or more millimeters. They are very conspicuous, on account of their opaque-white or yellowish color, in sharp contrast to the translucent golden yellow of the fat in which they occur. These areas are not infrequently surrounded by a narrow hemorrhagic zone. Such foci are most numerous upon the peritoneal surface, usually extending only a short distance below it, but also occur embedded in the fat. Particularly in the neighborhood of the pancreas, where they are most numerous, foci of necrosis may be large and confluent, giving a tallow-like whiteness to extensive areas of fat. Discrete foci in greater or less number may stud the perinephritic fat, the mesocolon, the mesentery, and the omentum. Foci of necrosis occur less frequently below the parietal peritoneum and upon the surface of the diaphragm.

In some instances fat necrosis has extended beyond the peritoneal cavity. Hansemann has twice seen foci of necrosis in the subcutaneous fat, while in several instances the fat of the pericardial and pleural cavities has been implicated to a slight extent. Chiari has described a remarkable instance of widespread fat necrosis. Gangrenous pancreatitis had caused such complete disintegration of the gland that the duct of Wirsung was eroded and communicated directly with the cavity of the bursa omentalis; areas of the fat necrosis, often the size of a pea, were present about the lesser peritoneal cavity, in the mesentery, in the subperitoneal fat of the abdominal wall, in the subpericardial and subpleural fat, and in the subcutaneous tissue.

¹ *Berliner klin. Woch.*, 1902, xxxix, 734.

Experiments by the writer have reproduced in cats the same extensive necrosis and show that it is the result of conditions which facilitate the penetration of pancreatic secretion into the tissues about the organ. When both pancreatic ducts in the cat were ligated, death, with necrosis of almost the entire abdominal fat, occurred within several weeks, being caused by pancreatic secretion dammed back upon the gland; foci of necrosis occurred outside the abdominal cavity, particularly in the fat of the pericardium. When pilocarpine muriate, which stimulates pancreatic secretion, was administered to an animal of which the pancreatic ducts had been ligated, death occurred in four days, and equally widespread necrosis of fat was found.

Microscopic examination of the foci of necrosis shows that the cells, of which the outlines are still preserved, although the nuclei have disappeared, contain at an early stage crystals of fatty acid. Calcium salts combine with these fatty acids to form calcium salts readily demonstrable by microchemical reactions. A few polynuclear leukocytes find their way into the necrotic tissue, but when numerous their presence is due to secondary infection. According to Wells the lesion experimentally produced may be recognizable three hours after the application of pancreatin to fat.

Proliferation of fixed tissue cells occurs in the periphery of the necrotic area. The lesions may so completely disappear that its site is no longer recognizable. Wells found fat necrosis in a dog of which the abdomen was opened four days after the injection of pancreatin, but seven days later the lesion had completely disappeared. In a case of acute cholecystitis Körte found at operation typical areas of fat necrosis; the patient recovered, but at a second operation, undertaken a year later, for the relief of symptoms suggesting the presence of a gallstone in the common bile duct, foci of necrosis were no longer present.

Clinical Significance of Fat Necrosis.—The facts already cited demonstrate that the lesion is caused by pancreatic juice diverted from its proper channels into the tissues about the gland. When the outflow of bile is obstructed, the bile dammed back upon the liver reaches the interstitial tissue of the gland, and hence is transported to all parts of the body. When the secretion of the pancreas, likewise dammed back upon the gland, reaches the interstitial tissue of the organ, and hence, probably by way of the lymphatic spaces, finds its way into tissues at some distance from the gland, its presence is not indicated by any colored constituent; as yet, moreover, there is no certain means of identifying constituents of the pancreatic juice in the urine. Focal fat necrosis is in such case the only readily recognizable effect of the aberrant secretion, and for diagnosis of pancreatic disease is available only to the surgeon who opens the abdomen. Hansemann has mentioned a unique case, in which circumscribed areas of cutaneous injections marked the site of foci of necrosis, shown by postmortem examination to be situated in the subcutaneous tissue of the abdominal wall, but rarely does the characteristic lesion implicate fat outside of the abdominal cavity.

Since disseminated foci of fat necrosis indicate in most instances acute hemorrhagic or gangrenous pancreatitis, its recognition at operation is of great importance. The conspicuous opaque-white areas studding the translucent yellow fat are unlikely to be overlooked when the omentum is exposed. By those who have not previously seen the lesion it may be mistaken for caseous miliary tubercles or for carcinomatous nodules which have undergone

necrosis, but the absence of elevation or other evidence of newly formed tissue shows that necrosis has affected the fat.

Since abundant evidence has shown that fat necrosis is due to the action of the fat-splitting ferment secreted by the pancreas, the possibility suggests itself that the ferment which is free in the tissues may be excreted by the kidneys. Recognition in the urine of a fat-splitting ferment derived from the pancreas would afford means for the diagnosis of obscure pancreatic disease accompanied by fat necrosis. With the urine removed at autopsy from the bladder of an individual dead with acute hemorrhagic pancreatitis and fat necrosis, the writer obtained a reaction which indicated the presence of the fat-splitting ferment. The method of identifying this enzyme was that described by Kastle and Loevenhart, who showed that the presence of fat-splitting ferment may be demonstrated by use of ethyl butyrate. Experiments of Hewlett¹ show that a variety of injuries to the pancreas of dogs are followed by the appearance in the urine of a ferment which is capable of decomposing ethyl butyrate into butyric acid and alcohol. Very little if any of such ferment is present in the normal urine; after obstruction of the pancreatic duct he found it during a period of from three to five days, but it is present in greatest amount as the result of experimental hemorrhagic pancreatitis. The method which Hewlitt describes in detail can be used to examine the urine when pancreatic disease with fat necrosis is suspected.

ACUTE HEMORRHAGIC PANCREATITIS.

In his monograph, published in 1842, Claessen collected cases which he regarded as examples of acute pancreatitis, and mentioned certain ill-defined symptoms of the disease, such as deep-seated epigastric pain, forcible vomiting of greenish material, faintness, thirst, and constipation. Klebs, in 1870, carefully described the anatomical changes which occur in the organ, and showed that pancreatic hemorrhage is associated with severe symptoms and sudden death, but did not distinguish between hemorrhage into the pancreas and hemorrhagic inflammation. He states that purulent peripancreatitis may be the result of hemorrhagic pancreatitis. Spiess had previously described a case in which sudden death was due to hemorrhage into this organ, and Rokitansky had mentioned the occurrence of acute hemorrhagic pancreatitis.

Friedreich (1875) based his description of acute primary pancreatitis upon 4 cases, 2 of which (Loeschner, Oppolzer) were hemorrhagic in character, while in a third case, observed by himself, the gland was the seat of multiple abscesses. In consequence of the small number of available cases, he was unable to give either completeness or precision to his description of the symptomatology of the condition. As acute secondary pancreatitis he designates parenchymatous degeneration, which is analogous to parenchymatous degeneration of the liver, kidneys, and other organs, and frequently occurs with acute infectious diseases; a second form of acute secondary pancreatitis which he mentions is the rare metastatic abscess which can occur in association with pyæmia. As instances of hemorrhage into the pancreas Friedreich described the case of Klebs and three described

¹ *Journal of Medical Research*, 1904, xi, 377.

by Zenker in the previous year. The cases of Zenker, in which in individuals dying almost immediately after the onset of symptoms hemorrhage into and about the pancreas was the only lesion found at autopsy, are examples of what has been subsequently designated pancreatic apoplexy. Zenker has pointed out that the amount of hemorrhage in these cases is too small to explain the sudden death, and referring to the experiments of Goltz, in which diastolic arrest of the heart beat is produced in frogs by tapping the abdomen, suggests that death is referable to an injurious influence upon the nervous system; Friedreich, however, ascribed death to pressure upon the solar plexus and semilunar ganglion brought about by sudden swelling of the pancreas.

The Middleton-Goldsmith Lecture of Fitz, published in 1889, contains a classification of acute pancreatic diseases which has served as a basis for the first accurate description of their symptomatology. He found that acute inflammation of the gland may be hemorrhagic, gangrenous, or suppurative. His description of the symptomatology of acute hemorrhagic pancreatitis has made typical instances of the condition recognizable during life. Gangrenous pancreatitis, according to Fitz, although it may follow other conditions, is usually the result of the hemorrhagic lesion. Suppurative pancreatitis resembles suppurative inflammation of other organs. Fitz noted the relationship of so-called fat necroses to acute pancreatic disease, already suggested by Chiari, and found that it more frequently accompanied hemorrhagic and gangrenous than suppurative inflammation.

Pancreatic Hemorrhage.—Those who have systematically described diseases of the pancreas have given special consideration to so-called *pancreatic apoplexy* or hemorrhage into the pancreas; the condition has medico-legal interest, since it is believed to be one of the causes of sudden unexplained death. Hemorrhage occurring into the substance of the gland may be caused by traumatism, or by malignant growth, and minute extravasations may be associated with various infectious diseases; such hemorrhage presents nothing peculiar to the organ. The literature of pancreatic disease contains numerous cases in which a hemorrhagic lesion of the gland has been the only explanation of the rapidly fatal illness of an apparently healthy individual. In some cases cited as examples of pancreatic hemorrhage, as Seitz points out, it is improbable that the pancreatic lesion has been the cause, whereas, in other cases, such as those described by Reubold and Rehm, after poisoning with morphine, after strangling, or after hemorrhage from the femoral vein, the amount of blood in and about the gland has been so small that it may be referred to the hemorrhagic extravasation which Chiari has described in association with postmortem or agonal self-digestion of the pancreas.

Numerous cases have demonstrated that extensive hemorrhage into the peripancreatic tissues may occur with acute hemorrhagic pancreatitis; necrosis and inflammation are primary, and the hemorrhage is their consequence. When both inflammation and hemorrhage co-exist, the difficulty of deciding which preceded the other is obviously great, for an accumulation of blood in and about the duodenum would quickly become the seat of bacterial infection, and an abscess cavity would be formed. The existence of hemorrhage without evidence of inflammation has been believed to demonstrate the occurrence of so-called apoplexy. Atheroma of arteries and fatty degeneration of the pancreatic parenchyma, cited as causes of hemorrhage, have been found in only a limited number of cases. Knowledge of the

pathology of acute hemorrhagic pancreatitis, recently acquired, has facilitated the description of such cases, and the literature of the last few years contains few, if any, instances of so-called apoplexy. One of the most typical of the recorded cases of pancreatic hemorrhage is described by Simpson. The pancreas was the seat of hemorrhagic infiltration, the organ was surrounded by semifluid clotted blood, and the intima of the splenic artery was apparently necrotic, although it is not stated that rupture of this vessel had occurred. In view of subsequent observations, which will be described, the presence of a gallstone lodged at the orifice of the common bile duct leaves little doubt that this case is a typical example of acute hemorrhagic pancreatitis.

Classification.—The classification of acute pancreatitis as hemorrhagic, gangrenous, and suppurative has been adopted by the majority of clinicians. Mayo-Robson describes pancreatitis under the headings: (1) acute, including cases in which death occurs within forty-eight hours of the onset; (2) subacute, including cases in which death does not occur for several weeks after onset; and (3) chronic, where the attacks are long continued or repeated and chronic inflammation or cirrhotic change is found in the pancreas. Mikulicz-Radecki,¹ who adopts the classification of Mayo-Robson, says of the three types of pancreatitis described by Fitz: "These three forms of pancreatitis, easily differentiated as they are in typical cases, merge into one another in most instances, or follow each other in succession." The same objection, perhaps in greater degree, is applicable to the classification of pancreatitis as acute, subacute, and chronic, for it groups together distinct morbid entities because under certain circumstances they exhibit similar symptoms. In the present description of acute pancreatic disease, hemorrhage into the pancreas or pancreatic apoplexy has been omitted, for reasons which have already been mentioned. Gangrenous pancreatitis has been described as a late stage of hemorrhagic pancreatitis, which usually precedes it, because there is little, if any, evidence that the lesion can occur as a primary change in the gland. At the same time, it is recognized that suppurative pancreatitis or peripancreatitis is in many cases associated with the gangrenous lesion.

Etiology.—Acute hemorrhagic pancreatitis occurs more frequently in men than in women. Among 41 instances of the hemorrhagic lesion, Körte found 37 were in males and 4 in females; in 40 cases of gangrenous pancreatitis, 21 were in males and 19 in females. The disease, therefore, appears to have a more rapidly fatal course in men than in women. Of 121 cases of hemorrhagic and gangrenous pancreatitis cited by Peiser,² 79 were in males and 42 in females. The greater number of cases occur between the ages of twenty and fifty years.

A variety of conditions has been believed to favor the onset of the disease. Individuals in apparently good health are not infrequently attacked; those with abundant adipose tissue are said to be especially susceptible. About one-half of the cases both of hemorrhagic and of gangrenous pancreatitis, Fitz found, are preceded by attacks of indigestion, accompanied by pain either referred to the region of the stomach or believed to be biliary colic. In a few instances anatomical evidence of *gastritis* and *duodenitis* has been obtained, but its etiological relation is doubtful. In two cases Simon and

¹ *Transactions of the Congress of American Physicians and Surgeons*, 1903, vi, 55.

² *Deutsche Zeit. f. Chir.*, lxx.

Stanley found intense gastroduodenitis with cellular infiltration of the duodenal submucosa, and since the duct of Wirsung was inflamed, they thought that the pancreatic lesion was the result of ascending inflammation. In one of these cases there was jaundice, and a calculus was found in the gall-bladder; the passage of a gallstone may perhaps explain the occurrence of the lesion of the pancreas. In a case of Reynold and Moore there was gangrenous duodenitis, and in six cases cited by Gessner there was severe gastritis. It is not improbable that such lesions may be secondary to the pancreatic disease. It has been suggested by Peiser, who has described a case in which hemorrhagic pancreatitis occurred two and a half weeks after *childbirth*, that pregnancy may favor its onset. Of the 8 cases which he cites, 5 occurred from five weeks to five months after labor and the relationship is doubtful.

From the hemorrhagic pancreas and from the associated foci of fat necrosis, a variety of *bacteria*, in many instances *Bacillus coli*, has been isolated, but according to almost all observers the results of bacteriological examination, which have been very inconstant, indicate that the various organisms which have been isolated bear no etiological relation to the disease and are secondary invaders of the injured tissue. The acutely inflamed gland may contain no bacteria.

Experimental Pancreatitis.—Experiments of Hlava have shown that the lesion of acute hemorrhagic pancreatitis can be reproduced by injecting artificial gastric juice into the pancreatic ducts of dogs. A similar lesion which has followed the introduction of cultures of *Bacillus coli*, *Bacillus lactis aërogenes*, and *Bacillus capsulatus* of Friedlander, the same writer has attributed to the acid products of these organisms. Carnot reproduced the lesion by injecting diphtheria toxin, and Flexner, in a large series of successful experiments, used weak solutions of hydrochloric, nitric, and chromic acids, sodium hydroxide, and formalin. The suggestion of Hlava that gastric juice may be driven by antiperistaltic action of the intestine into the pancreatic ducts is not supported by any evidence.

Traumatic Pancreatitis.—Hemorrhagic and gangrenous pancreatitis with disseminated fat necrosis has in a few instances so directly followed abdominal injury that the relation to traumatism cannot be doubted. Injury to the pancreas is usually associated with such severe damage to adjacent structures that a fatal result is rarely referable to the gland. Incisions into the pancreas of animals heal readily, while in 7 of 12 cases cited by Mikulicz-Radecki recovery occurred, although operation had demonstrated a wound of the pancreas. In a small number of cases hemorrhagic and gangrenous pancreatitis has followed blows upon the epigastric region which have not produced superficial wounds; in such cases injury has been due to the kick of a horse (Selberg), crushing between the buffers of railroad cars (Schmidt), a violent fall (Hahn). A typical case is described by Selberg. A man, aged thirty-eight years, was kicked by a horse in the region of the stomach, and was for a time unconscious. There was pain and tenderness in the epigastric region, with gradual distention and vomiting. The temperature remained normal. At autopsy there was generalized fibrino-purulent peritonitis. The omentum was studded with foci of fat necrosis; the head of the pancreas was wholly necrotic, and the remainder of the organ was infiltrated with blood.

Little is known concerning the pathogenesis of traumatic gangrene of the

pancreas. Thrombosis of bloodvessels may perhaps explain the lesion. Repeating the experiments of Hildebrand and Dettmer and subsequent observers, Doberauer has shown that obstruction to the circulation, together with other injury to the gland, may be followed by hemorrhagic necrosis of the pancreas and fat necrosis.

Cholelithiasis.—Those who have described acute hemorrhagic pancreatitis have noted that it is not infrequently associated with cholelithiasis. In 1903 it was possible to collect from the literature 37 cases in which hemorrhagic or gangrenous pancreatitis was associated with gallstones, and at least 9 similar cases have since been described. In 9 cases of the hemorrhagic lesion a calculus has been lodged near the termination of the bile duct, and one case examined by the writer¹ in the pathological laboratory of the Johns Hopkins Hospital disclosed a mechanism by which a biliary calculus can produce the lesion. A corpulent man, aged forty-eight years, who had suffered with attacks of indigestion, was suddenly seized with severe pain in the abdomen, accompanied by nausea. The pain diminished in severity, but two days before his death returned with its former intensity. At autopsy widely disseminated fat necrosis was found, and the pancreas, which was much enlarged, had an almost uniform reddish-black color. A small gallstone had lodged at the orifice of the diverticulum of Vater, so that it had converted the common bile duct and the duct of Wirsung into a continuous closed channel, from which neither bile nor pancreatic juice could escape. Bile had penetrated into the pancreatic duct, doubtless injected by the gall-bladder. Bacteriological examination of the pancreas gave negative results. Bunting² has described a case almost identical with that just cited. Experiments on dogs demonstrated that the bile of one animal injected into the pancreatic duct of a second is capable of causing typical hemorrhagic pancreatitis, which is fatal often within twenty-four hours, and like the human lesion is characterized by necrosis of the parenchyma, hemorrhage, and inflammatory reaction.

No data at present available indicate with how great frequency acute hemorrhagic pancreatitis is caused by gallstones. In 4 of 5 cases in the Johns Hopkins Hospital cholelithiasis has been present, and in 2 of these cases calculi have been found lodged at the orifice of the common bile duct. In many cases doubtless gallstones have been overlooked at autopsy. It is noteworthy that the gallstones usually found in the bile passages and in the gall-bladder in these cases have been of such small size that they might readily occlude the duodenal orifice of the diverticulum of Vater without closing the communication between the common bile duct and duct of Wirsung. In only a limited number of individuals is the anatomical structure of the diverticulum of Vater such that a calculus could convert the two ducts into a continuous closed channel. In 1 of 10 individuals the two ducts have no common channel, but open separately at the summit of the bile papilla, and in only 32 of 100 normal specimens which have been examined was the diverticulum of Vater of such size that a small calculus might occlude its orifice without completely filling it and thus obstructing one or both ducts which enter it.

Special Pathology.—The appearance of the pancreas varies with the duration of the disease. In instances in which death has occurred within

¹ *Johns Hopkins Hospital Bulletin*, 1901, xiv, 182.

² *Ibid.*, 1906, xvii, 265.

forty-eight hours after the onset of symptoms the organ is much enlarged, and in swelling assumes an irregularly cylindrical shape. The areolar tissue in the neighborhood of the gland may be infiltrated with blood and the lesser peritoneal cavity usually contains blood or blood-stained fluid. Opaque, white foci of fat necrosis studding the retroperitoneal fat, transverse mesocolon, perhaps the mesentery elsewhere, and the subperitoneal tissue of the abdominal walls, are abundant, particularly in the immediate neighborhood of the gland. The tissue of the pancreas is firm in consistence and has in large part assumed a dark-red, reddish-brown, or even reddish-black color, and not infrequently presents a mottled or marbled appearance due to the presence of sharply defined areas of yellowish white, relatively normal parenchyma. The proportion of transformed parenchyma varies considerably in different cases, but of importance is the fact that rarely, if ever, is the entire parenchyma affected by the lesion.

Characteristic of the lesion is the widespread necrosis which affects secreting parenchyma, interstitial tissue, and the walls of bloodvessels. Histological examination shows that cells are transformed into homogeneous structures without nuclei. The architecture of the tissue may be preserved, but outlines of acini and fibrous tissue are often completely lost. Transition from such areas of complete death of tissue to normal parenchyma is abrupt and marked by a zone containing fragments of nuclei, polynuclear leukocytes, red blood corpuscles, and fibrin. The interstitial tissue of the parenchyma which is still intact is infiltrated with blood, while in the necrotic and partially disintegrated areas occur larger collections of much changed blood. At an early stage of the disease the inflammatory nature of the lesion is well shown at the margin of the relatively normal tissue, where inflammatory products, particularly leukocytes and fibrin, are found in great abundance.

The pathogenesis of the condition is best studied in the experimental lesion, which, as already stated, does not differ in gross and histological characters from that which occurs in human cases. Bile and a variety of irritant substances coming into contact with cells of the secreting parenchyma cause their death, and this necrosis is associated with such injury to the walls of bloodvessels that red blood corpuscles escape in great quantity. Hemorrhage may be the result of necrosis before inflammatory changes are well marked, but very quickly evidence of inflammation is found at the margin of relatively intact parenchyma. Flexner and Pearce have shown that degeneration, hemorrhage, and emigration of leukocytes occur within from one to two hours after the injection of artificial gastric juice into the pancreatic duct of dogs. There is probably much truth in the suggestion of Klebs that hemorrhage is the result of corrosive action of the pancreatic juice. The various irritant substances which produce the lesion acting injuriously upon the tissue of the organ not improbably subject it to the action of its own secretion.

Gangrenous Pancreatitis.—Fitz showed that acute hemorrhagic pancreatitis may terminate, in the condition known as gangrene and in at least half of the recorded cases of what he designated gangrenous pancreatitis there was evidence of hemorrhage. In cases of hemorrhagic pancreatitis which have survived the onset of symptoms a week or more the gland assumes a dark-red, reddish-black, or black color, and the tissue is dry and covered by changed blood. At the end of the second week the organ may form a soft,

black, friable mass, while the lesser omental cavity contains chocolate-colored fluid in which are bluish-black spots. Yellowish spots of softening may occur in the pancreas and the gland may be finally transformed into a soft mass attached by only a few shreds of tissue to the abdominal wall, or, having undergone complete sequestration, according to the description of some observers may lie free in the omental cavity. In such cases the lesser peritoneal cavity is distended with dark bloody or blackish, often foul-smelling, fluid containing particles of necrotic tissue. Infection with a variety of bacteria has occurred, and the abscess cavity thus formed is limited by the lesser peritoneal cavity; the foramen of Winslow is closed by adhesions, and there is in most instances no general peritonitis. The fat about the pancreas and in the wall of the lesser peritoneum is the seat of confluent areas of fat necrosis which as the result of softening and erosion produce an irregular ragged surface.

In some instances an extension of the abscess cavity forms a pocket in the retroperitoneal tissue over the left kidney. Perforations, usually with ragged frayed edges, may be found in the wall of the stomach or duodenum, doubtless caused by penetration of the abscess into the viscus. In a case described by Chiari there were two perforations into the stomach and five into the jejunum. In a recorded case by Chiari part of the gangrenous pancreas, identified by Rokitsky, was passed from the rectum.

No sharp distinction can be made between hemorrhagic and gangrenous pancreatitis. Histological and experimental study has shown that widespread death of tissue is an essential feature of the hemorrhagic lesion, and in experiments on animals is demonstrable within a few hours after the injection of an irritant substance. Subsequent changes occurring in the extravasated blood acted upon by the pancreatic juice and by bacteria which have invaded the dead tissue cause such discoloration and disintegration of the affected gland that it is described as gangrenous. Suppuration is always secondary to the hemorrhagic necrosis and is not its cause.

When, with the experimental lesion, recovery ensues, active proliferation of connective tissue occurs and the newly formed tissue invades and in part or wholly replaces the necrotic parenchyma. Evidence of such reparative changes have been found in patients who have survived the severity of onset, and is doubtless present in the comparatively small number of instances in which complete recovery occurs.

Symptoms.—Fitz carefully defined the symptoms of hemorrhagic and of gangrenous pancreatitis, and although he recognized that many cases showed varying combinations of the lesions thus designated, described them separately. Most writers who have subsequently described instances of either condition have adopted the classification of Fitz and have used that designation which more accurately describes the anatomical changes which have been found at autopsy.

Course of the Disease.—In those cases in which death occurs within a few days after the onset the hemorrhagic character of the lesion is predominant, and those cases which pursue a rapidly fatal course have been described as hemorrhagic pancreatitis. The relation between the duration of the disease and the character of the changes found after death varies considerably, but toward the end of the second week, and in many instances earlier, the pancreas has assumed the appearance described as gangrenous, and those

cases have been described as gangrenous pancreatitis in which either the initial symptoms of shock have not been fatal or the onset has been unusually mild. Since the disease apparently pursues a more violent course in men than in women, the hemorrhagic lesion has been more frequently encountered at autopsy in men. When the disease has assumed the subacute course which characterizes most cases of what has been called gangrenous pancreatitis, symptoms due to suppuration and accumulation of exudate in the lesser peritoneal cavity make their appearance.

Stage of Hemorrhagic Inflammation.—Characteristic of the acute disease is its sudden onset, the severity of the pain which is localized in the epigastric region, and the associated symptoms of shock and intense depression of the circulatory system. One group of cases described by Nimier as *foudroyant* terminate fatally within a few hours. Such cases have been described as instances of pancreatic hemorrhage or apoplexy. In a large proportion death occurs within the first four or five days, while about half of the cases pursue a subacute course, death occurring after several weeks or months.

The disease in many instances affects individuals who have suffered with repeated attacks of abdominal pain, but in many cases the patients have had good health, robust men often somewhat corpulent being not infrequently affected. *The preceding attacks of pain* are of two kinds, being referable either to the gastro-intestinal tract or to the bile passages. In nearly one-half of the cases collected by Fitz there had been attacks of indigestion, with pain at times of great severity, nausea, and vomiting. He regarded the previous digestive disturbance as gastric or gastroduodenal rather than enteric. In about one-fifth of the recorded cases, according to Gessner,¹ there have been, perhaps for several years, typical attacks of gallstone colic often accompanied by jaundice, until finally a similar attack of unusual severity is accompanied by symptoms of collapse.

The pain of onset, which appears with surprising suddenness, is of great violence and is localized above the umbilicus; it may be constant or occur in paroxysms, and in the acutely fatal cases persists until death. The pain has been occasionally described as present above the umbilicus and to the left of the median line following the course of the pancreas, but the seat of greatest intensity may be the left or right hypochondrium, or even the lower abdominal region. The epigastric region is sensitive to pressure, and there is usually rigidity of the abdominal muscles. Intense pain in the back radiating toward the legs has been described.

The appearance of pain is rapidly followed by *vomiting*, rarely by nausea alone. Vomiting may recur at short intervals or may stop for several hours or even several days, to be repeated with perhaps equal severity. The vomitus is usually copious and bile-stained, but may be mucous, and in several instances has been dark brown or bloody.

Symptoms of shock accompany the pain and vomiting of onset and accompanying exacerbations of the symptoms usually precede death. There is profound weakness; the pulse is accelerated, and in several instances cyanosis has been a conspicuous symptom. Experiments of Gulecke,² Doberauer,³

¹ *Deutsche Zeit. f. Chir.*, 1899, xliv, 65.

² *Arbeiten a. d. Chir. Klinik. d. Königl. Univ. Berlin*, 1906, xviii, 368.

³ *Beiträge zur klin. Chir.*, 1906, xlviii, 465.

and Egdahl¹ indicate that substances perhaps formed by autodigestion of pancreatic tissue depress the circulation and cause profound poisoning.

Although the abdomen is rigid and often distended, particularly in the epigastric region, a *tumor mass* referable to the pancreas is rarely if ever palpable.

In about one-half of the cases there is *constipation*, but in a considerable number of cases *diarrhoea* is present from the beginning or may follow constipation. The occurrence of constipation in association with the severe symptoms of onset has made the diagnosis of intestinal obstruction in such cases frequent, and the nature of the disease has been determined at operation undertaken with the purpose of relieving obstruction. Gessner states that passage of faeces occurs spontaneously or as the result of enemata in many cases on the fourth day. In six cases collected by this author the enlarged head of the pancreas was found either at autopsy or at operation to encroach upon the lumen of the duodenum, but in most cases no anatomical evidence of obstruction has explained the symptoms of intestinal obstruction. It has been suggested that irritation of the solar plexus by hemorrhage or by pressure may favor the occurrence of constipation, since stimulation of the splanchnic nerves inhibits intestinal movements. The stools are not infrequently clay-colored; in only one case (Chantemesse and Griffon) fatty stools were present, although death occurred two days after the onset of symptoms. The faeces rarely contain blood.

Jaundice, varying in intensity, was present in 10 per cent. of the cases collected by Gessner, who has given special attention to the relation between acute pancreatitis and disease of the biliary passages.

Fever is rarely present during the early stage of the disease, and with symptoms of collapse the temperature may be subnormal. Elevation of temperature with chills has been observed.

Leukocytosis has been found in cases in which the blood has been examined. In a case of Hunt² the leukocytes numbered 37,000 per cmm. on the first day of the disease. In a case of Sandler their number was not increased.

Glycosuria has occurred in only a small proportion of cases of hemorrhagic pancreatitis (Benda and Stadelmann, Cutler, Sarfert and Simmonds). The case of Benda and Stadelmann is noteworthy, because the symptoms were those of rapidly fatal diabetic coma. The patient became comatose soon after the onset of violent abdominal pain; 3.4 per cent. of sugar was found in the urine. The pancreas, with the exception of a part of the head, formed a soft bloody mass.

Stage of Gangrene.—Those cases which have survived the early stage of the disease have been described as instances of gangrenous pancreatitis. At the end of several days—Gessner regards the fourth day as critical—the symptoms of onset may diminish in violence; pain and tenderness in the epigastric region are still present, or may recur at intervals, and is not infrequently associated with vomiting. Constipation, if present, disappears and may be followed by diarrhoea. Symptoms which indicate the presence of so-called gangrene, with bacterial invasion of the necrotic gland and adjacent peritoneal surfaces, are fever and epigastric tumor. The transition to the

¹ *Journal of Experimental Medicine*, 1907, ix, 385.

² *Boston Medical and Surgical Journal*, 1905.

stage of necrosis and suppuration usually occurs toward the end of the second week.

The *temperature*, which during the early stage of the disease shows no elevation, is moderately increased and irregular. Although usually little more than 100°, it may reach 104° F., and in some instances there are repeated chills. The disease may, however, pursue its course without fever. Leukocytosis was present in two cases of gangrenous pancreatitis described by Thayer. In one case the leukocytes on the nineteenth day numbered 18,300; in the second case during the fourth week of the disease they numbered 33,700, although the temperature at the same time varied from 97.6° to 98.6° F.

The *tumor mass*, due in great part at least to accumulation of exudate in the lesser peritoneal cavity, usually appears in the epigastric region extending toward the spleen, and varies considerably in size and distinctness. It has been described at times as a rounded mass the size of a child's head situated above the umbilicus, while in other cases there has been an ill-defined resistance in the same region. In all of 5 cases of gangrenous pancreatitis Körte found a tumor mass which in 3 instances was first found on the left side in the region of the left hypochondrium and flank, subsequently becoming palpable between stomach and colon. In 5 cases described by Thayer a tumor mass was found in the epigastric region; in 2 cases pancreatic gangrene was demonstrated by autopsy, while in 3 recovery followed operation and the nature of the lesion was not clearly defined.

In stout individuals palpation may be difficult, but is facilitated by the emaciation which usually occurs with long continuance of the disease. The muscular spasm which hinders palpation during the acute stage of the disease disappears. The lower border of the tumor mass may descend with respiration; occasionally pulsation is transmitted from the aorta, and has been found to disappear in the knee-chest position. Inflation of the stomach and perhaps of the colon may aid in the localization of the tumor mass. Tympany of the stomach separates it from the liver, and after inflation of the viscus tends to cover it. The colon lies along the lower border of a tumor mass due to an exudate within the lesser peritoneal cavity. When the abscess cavity eroding the tissue over the left kidney tends to make its way toward the left lumbar region, resistance may be felt below the left costal margin extending downward as far as the rim of the pelvis.

The stools, which are not infrequently diarrhoeal in a few instances, have contained blood. Steatorrhœa has rarely if ever been observed, but in a case of von Noorden's chemical examination showed that the proportion of split fat in the faeces was greatly diminished. In 2 cases described by Chiari recovery followed the expulsion from the rectum of necrotic pancreatic tissue.

Jaundice may occur, and was present in one-fifth of the cases of gangrenous pancreatitis collected by Fitz.

Glycosuria is an infrequent symptom of pancreatic necrosis. In the cases of Israel and of Fleiner the disease occurred in individuals who had previously suffered from diabetes. In the case of Middleton sugar was found in the urine removed from the bladder at autopsy. In the case of Brentano 6 per cent. of sugar was found in the urine ten days after operation and four days after the discharge from the wound of a mass of necrotic tissue 12 cm. in length; previous examination of the urine had not been

made. Peiser found sugar in the urine two months after the onset of symptoms; it is probable that almost the entire gland was affected, since a few days after the operation a sequestrum of pancreatic tissue 19 cm. in length was discharged from the wound.

Complications and Sequelæ.—The most important complications, namely cholelithiasis and suppurative peritonitis affecting the lesser peritoneal cavity, have been described. Erosion of the tissue over the left kidney and sinking of the bursal abscess toward the left lumbar region has also been mentioned. Perforation of the stomach, duodenum, and transverse colon occur not infrequently, and in a case described by Thayer the three organs were the seat of erosion. Blood or foetid pus rarely appear in the feces as the result of such accidents.

Within the necrotic tissue of the pancreas, bloodvessels become the seat of thrombi which not infrequently extend to adjacent vascular trunks. The splenic vein may be occluded and in a case described by Fitz-thrombosis of the splenic vein was present in a patient who died ten days after the onset of symptoms.

Diabetes is infrequently associated with pancreatic gangrene, but may occur either as a complication or a sequel. Such instances have already been cited. In a case of Körte 2 per cent. of sugar was found in the urine twenty months after an operation at which necrotic pancreatic tissue had been removed. Sugar was not present before or immediately after operation; seven years after operation the patient was living, but much emaciated as the result of diabetes.

An unusual accident has been described by Langton. Two years after an operation, at which bloody fluid had been removed from the lesser peritoneal cavity, death occurred suddenly. The pancreas was in large part replaced by scar tissue; the scar was in contact with the portal vein from which fatal hemorrhage had occurred.

In a considerable number of cases pancreatic and peripancreatic *cysts* are preceded by acute symptoms which are similar to those of hemorrhagic pancreatitis, but in most instances it is impossible to determine by anatomical examination after a considerable lapse of time the character of the primary pancreatic lesion. A few cases indicate that peripancreatic cysts formed within the bursa omentalis or even pancreatic cysts may follow hemorrhagic pancreatitis. In a case described by Francke¹ intense pain associated with vomiting appeared with great suddenness; two weeks later a tumor was found in the left hypochondriac region and gradually increased in size. At operation, five weeks after onset, an elastic thin-walled cyst, the size of a man's head, occupied the lesser peritoneum and surrounded the pancreas, which was the seat of necrosis and old hemorrhage; foci of fat necrosis were present in the omentum and mesentery. In a similar case of Rasumosky a tumor mass in the epigastrium was noted five hours after the onset of symptoms, and at operation three weeks later was found to be due to a cyst in which necrotic pancreatic tissue was found. Adler² has described a so-called pseudocyst of the pancreas which was probably the result of acute hemorrhagic pancreatitis; the pancreas which formed part of the cyst wall was necrotic and the site of old hemorrhage. Frerichs and Finotti have

¹ *Deutsch. Zeit. f. Chir.*, 1900, liv, 399.

² *Virchow's Archiv*, 1904, clxxvii, Suppl. Heft 154.

described peripancreatic cysts which apparently followed hemorrhagic necrosis of the pancreas. When blood and necrotic pancreatic tissue remain sterile the digestive ferments of the pancreas may perhaps cause softening and encapsulation by fibrous tissue. The possibility that this process might take place wholly within the substance of the pancreas is suggested by a case of acute hemorrhagic pancreatitis with fat necrosis described by Dressel; a cavity filled with clotted blood the size of a child's head was present within the substance of the gland.

Diagnosis.—For the recognition of acute hemorrhagic pancreatitis the mode of onset is important, namely, its suddenness and violence with intense epigastric pain, vomiting, and symptoms of profound collapse. When acute hemorrhagic pancreatitis accompanied by constipation resembles intestinal obstruction, the epigastric localization of pain and distention, the intensity of the pain, and collapse point to pancreatic disease. Stercoraceous vomiting is rarely present, and the increase of peristalsis recognizable with intestinal obstruction by abdominal inspection and auscultation is not present.

Since the disease may be preceded by repeated attacks of typical biliary colic, and when caused by the lodgment of a small calculus in the diverticulum of Vater is associated with symptoms of gallstones, a differential diagnosis may be very difficult. When an *attack of biliary colic* is accompanied by pain of unusual intensity present throughout the epigastric region and perhaps more intense on the left side, especially when there are symptoms of sudden and profound collapse, hemorrhagic pancreatitis should be suspected. With *perforation of the gall-bladder* following cholecystitis the symptoms are usually localized on the right side.

When the disease has assumed a subacute course, the recognition of a tumor mass in the epigastric region between stomach and colon suggests the presence of an exudate within the bursa omentalis, but gives positive evidence of pancreatic gangrene only when its appearance follows the acute symptoms already described. Symptoms indicating disturbed pancreatic function such as glycosuria or steatorrhea are rarely present, but both in the acute and subacute stages of the disease accurate chemical methods applied to the examination of urine and feces may give additional information.

Ulcerative processes associated perhaps with malignant tumors of the stomach, duodenum, or transverse colon may produce *suppurative inflammation limited to the lesser peritoneal cavity*, particularly when they cause perforation. The suddenness of onset, the intensity of the pain, and the rapidity of collapse indicate the presence of acute hemorrhagic pancreatitis, especially when there is no history suggesting gastric or duodenal ulcer or other lesion which might cause perforation of the stomach, duodenum, or transverse colon.

Pulsation of a tumor caused by exudate in the lesser peritoneal cavity, being transmitted from the aorta, is not expansile and disappears when the patient rests upon his side or on knees and elbows; Langdon has described such a case simulating *ruptured aneurism*.

Treatment.—Although hemorrhagic pancreatitis cannot be directly influenced by any available means, in some instances, perhaps, the profound collapse of onset may be overcome, so that the disease may reach the subacute stage, in which it is more susceptible to successful surgical interference,

Intense epigastric pain usually requires morphine, but in several instances the violent pains of onset have been controllable only by inhalation of chloroform. For the vomiting Körte recommends the withholding of food and lavage of the stomach. Enemata of warm water injected into the colon may serve to overcome constipation and distention, while stimulating and nutrient enemata may serve to sustain the strength of the patient. With collapse indicated by a weak pulse and cyanosis, subcutaneous or intravenous infusions of normal salt solution may be employed.

The value of operation during the early stage, when symptoms of circulatory depression are predominant, has been much disputed. Loss of blood is rarely an important feature of the disease, and it is improbable that hemorrhage from the necrotic tissue could be controlled under the condition which such an operation would present. A mistaken diagnosis, usually of intestinal obstruction, has been the incentive for a majority of the operations undertaken at this stage, and the recognition of characteristic foci of disseminated fat necrosis in the great omentum has given the first indication that the pancreas has been diseased. Careful searching of the intestine drawn out through the abdominal wound, together with prolonged anaesthesia, has doubtless had an unfavorable result in many instances. Mikulicz-Radecki recommends evacuation of the hemorrhagic exudate contained in the lesser peritoneal cavity and free drainage of the omental bursa with gauze after exposure of the vault of the diaphragm by resection of the tenth and eleventh ribs, if necessary.

Statistics collected by Gessner indicate that operations undertaken up to the end of the second week after onset, and therefore during the stage of hemorrhagic inflammation, are much more frequently fatal than those undertaken at a later period when the disease has become subacute and so-called gangrene with peripancreatic abscess is present. Of 41 instances in which operation was performed upon patients suffering with the disease, 21 occurred during the first two weeks, and in only one case (Halsted) did recovery follow. In most instances a laparotomy in the middle line was performed, the nature of the disease was not determined, the abdomen was closed, and death followed within a few hours. Of 20 cases operated upon at a later period, when peripancreatic abscess had formed, 6 recovered. In the larger statistics of Mikulicz-Radecki, published about five years later (1903), there were 46 operations in the acute stage, with 9 recoveries, and 35 operations in the subacute stage, with 18 recoveries. This surgeon has been unwilling to attribute much significance to these figures, since the relative proportion of untreated cases which die in the first stage is unknown. He has maintained that early operation limits the extent of necrosis and sequestrum, and gives diminished opportunity for serious complications, such as venous thrombosis and pyæmia. Operation under local anaesthesia may diminish the tendency to shock. Hahn made an incision with the aid of local anaesthesia, and, finding fat necrosis, evacuated a large hemorrhagic exudate; the abdomen was drained.

Since the disease is frequently caused by gallstones, it is desirable to examine the biliary passages at operation, and to determine if calculi are present. The continued presence of a calculus at the duodenal orifice of the diverticulum of Vater may increase the severity of an existing lesion, and its removal is desirable. The calculus which causes the obstruction is usually of small size and not infrequently the gall-bladder has contained

a large number of minute calculi, which during expulsion can perhaps repeatedly divert bile into the pancreatic duct. In a patient operated upon ten days after the onset of symptoms Kelly found fat necrosis; he removed from the gall-bladder about fifty very small calculi of almost uniform size; the patient recovered. That the existence of diabetes mellitus should not necessarily prevent operation for pancreatic necrosis is indicated by a case described by Peiser. Before operation the urine contained 4 per cent. of sugar, acetone, and diacetic acid; the excretion of sugar was not influenced by the evacuation of a peripancreatic abscess, accomplished by two operations. The patient lived five months after operation, and death was the result of diabetes mellitus.

SUPPURATIVE PANCREATITIS.

Suppurative inflammation of the pancreas, characterized by accumulation of inflammatory products and softening of tissue, with consequent formation of one or more cavities containing purulent fluid, does not differ from similar changes in other organs. In many instances the disease, which is the result of bacterial invasion, occurs as a primary infection, but in other cases it follows acute hemorrhagic pancreatitis, the hemorrhagic and necrotic tissue which is characteristic of this disease being particularly susceptible to bacterial infection; hence the so-called gangrenous pancreas may contain abscess cavities, and in such instances a distinction between gangrene and suppuration is impossible. Moreover, acute inflammation of the pancreas, which has persisted for several weeks, almost constantly extends to the lesser peritoneal cavity, which with bacterial invasion becomes the seat of suppurative inflammation. In many cases described as instances of suppurative pancreatitis operation has disclosed an abscess in this region, but no subsequent postmortem examination has disclosed the exact anatomical condition; in reports of many such cases no attempt has been made to distinguish between pancreatic and peripancreatic abscess. With gangrenous pancreatic inflammation, symptoms of peripancreatic abscess may be more conspicuous than those referable to the gland itself, and the lesion found at autopsy several weeks after the onset of symptoms may present nothing characteristic of the hemorrhagic and gangrenous lesion.

Etiology.—When suppurative inflammation is not the result of simple extension from an adjacent organ, there are two possible paths of infection, namely, the bloodvessels and the ducts. Metastatic abscesses are rarely found in the pancreas. Infection by way of the bloodvessels may explain some cases of suppurative inflammation, but more readily demonstrable and probably of far greater frequency is ascending infection by way of the ducts. Experimental studies show that suppurative lesions may follow the injection of bacteria into the pancreatic ducts. Suppurative pancreatitis has been produced by Körte and by Carnot by injection of bacilli of the colon group into the duct.

Valve-like folds within the diverticulum of Vater prevent the entrance of the duodenal contents, so that after death it is impossible to force material from the intestine into the duct. The flow of secretion doubtless exerts a protective influence by washing foreign material from the duct. Obstruc-

tion of the duct of Wirsung favors the entrance of microorganisms, and many cases of pancreatic suppuration are associated with occlusion of the duct of Wirsung by gallstones lodged in the diverticulum of Vater, by pancreatic calculi, and by malignant growths compressing the duct. In a case described by Pearce multiple abscesses in the head of the pancreas followed obstruction of the duct by carcinoma of the diverticulum of Vater.

The probability of infection is much increased when the presence of gallstones has previously caused suppurative inflammation of the bile ducts. In two cases of Mayo Robson's, abscess of the pancreas accompanied cholelithiasis with suppurative cholangitis. In a case of suppurative pancreatitis recorded by Dieckhoff biliary calculi had actually found their way into the duct of Wirsung. In a case described by Fuchs¹ it is probable that pancreatic abscess had been secondary to localized hemorrhagic pancreatitis caused by a gallstone lodged in the diverticulum of Vater.

In the small number of cases in which bacteriological examination of the purulent fluid has been made, a variety of microorganisms has been found. In the case of Maas and of Etienne both cocci and bacilli were found, among which, organisms having the cultural characters of *Bacillus coli* were numerous. In one of the cases described by Fitz several varieties of bacilli and a staphylococcus were isolated. An organism resembling *Bacillus proteus* has been isolated (Hauser); Dieckhoff found diplococci resembling *Diplococcus pneumoniae* and in a case of Körte streptococci were found.

Special Pathology.—With acute inflammation of the pancreas there may be polynuclear leukocytes within and about the ducts of the gland, and the interstitial tissue may be distended by edema. Such inflammation, which doubtless begins as an ascending infection of the ducts does not necessarily proceed to suppuration, and may be unaccompanied by macroscopic change. It is, however, not improbable that similar infection of greater severity may cause diffuse suppuration in the organ.

In a case described by Etienne² the entire gland, which was enlarged, was infiltrated with thick, yellowish, purulent fluid, which collected upon the cut surface; drops of pus escaped from the incised ducts. In other instances destruction of tissue is more advanced and the organ contains communicating cavities of irregular shape filled with pus, or there may be numerous isolated abscesses. In some cases almost total destruction of tissue has been described, a large cavity filled with pus being found to occupy the site of the pancreas. In a case described by Paul such a cavity contained 600 grams of purulent fluid in which were fragments of pancreatic tissue; inflammation had doubtless extended to the lesser peritoneal cavity, which formed the site of the abscess described. Localized abscesses may be situated in any part of the gland, but are most frequent in the head.

The abscess cavity may be surrounded by a fibrous capsule. Fitz observed that fat necrosis occurs less frequently in association with suppurative inflammation than with hemorrhagic or gangrenous pancreatitis; foci of fat necrosis are found when purulent inflammation is the result of hemorrhagic pancreatitis.

Symptoms.—Since suppurative pancreatitis may occur as a complication of acute hemorrhagic pancreatitis, of lithiasis, of cyst, or of cancer,

¹ *Deutsch. med. Woch.*, 1902, xxviii, 829.

² *Arch. de méd. expér.*, 1898, x, 177.

while it is sometimes the result of ascending infection from the inflamed duodenum or bile passages, its mode of onset and symptomatology vary considerably. In one group, including more than one-half of the cases collected by Körte, the onset is sudden and associated with intense epigastric pain, vomiting, and collapse; the symptoms of onset do not differ from those of acute hemorrhagic pancreatitis, and it is not improbable that in a considerable proportion of these cases at least, infection and suppurative inflammation have followed hemorrhagic necrosis. The severity of the symptoms diminishes, and the disease in most instances pursues a chronic course, with exacerbations of pain and gastro-intestinal disturbance occurring at irregular intervals. In a smaller group the onset is gradual, and for a considerable time there is abdominal pain, or perhaps only a sense of discomfort in the epigastric region, together with gastric symptoms of variable intensity.

The tendency of suppurative pancreatitis to pursue a chronic course is shown by 14 cases cited by Fitz; 6 were fatal in the first month, 3 during the second month, and 5 between the fourth and eleventh months. In 22 of 46 cases collected by Körte death occurred between one month and one year after the onset of the disease. In a few cases death occurs more rapidly.

In most cases there is either diarrhœa or constipation, the former being present in considerably more than half of the cases. In the case described by Nicolas hemorrhage from the bowel occurred several times, and at autopsy an abscess in the pancreas was found to communicate by a fistulous opening with the duodenum. Hiccough was present in 5 cases collected by Étienne, in 2 of which it was incessant. In 2 of the cases peritonitis may have caused the symptoms.

Jaundice occurs in a considerable number of cases, and may be due to cholelithiasis, which has afforded conditions favoring infection of the pancreatic ducts. Jaundice may be caused by an abscess, situated in the head of the gland and compressing the bile ducts.

Symptoms indicating the occurrence of suppuration are fever and abdominal tumor. In the greater number of cases there is elevation of temperature, which is moderate in degree, but in some instances the temperature has been normal or even subnormal. In one group of cases, according to Fitz, the onset is violent and there are frequent chills and irregular, often high, temperature, exceeding 105° F.; chills may occur at the onset and recur during the course of the disease. In another group of cases the early symptoms present little that suggests the presence of suppuration; there is loss of appetite, progressive weakness and emaciation, perhaps diarrhœa or jaundice, but no pain or fever. Leukocytosis has been present in cases in which purulent fluid has been removed from the lesser peritoneal cavity (Thayer, Murray).

A more or less clearly definable tumor mass occupying the position of the pancreas has been noted in hardly more than a fourth of the recorded cases (Körte), but when present this symptom is significant for both diagnosis and operative treatment. In many cases there is abdominal distention not infrequently localized in the epigastric region, and pain and tenderness in the same situation may further prevent satisfactory palpation, which may be more readily performed after the subsidence of the violent symptoms of onset. The detection of deep-seated resistance behind the

stomach or between stomach and colon, absent perhaps during the early stage of the illness, may first suggest that the pancreas is the seat of disease.

Disturbances of pancreatic function, although significant when present, are not commonly associated with pancreatic abscess, which in most instances leaves relatively normal a considerable part of the gland. Glycosuria has been present in a small number of cases. In the case of Nicolas diabetes was doubtless the result of pancreatic lithiasis, which had brought about conditions favorable for abscess formation. In the cases of Hartley and Atkinson almost complete destruction of the gland was associated with glycosuria; Frison and Frerichs found sugar in the urine. In the case described by Hartley fat discharged with the fæces resembled cod-liver oil, and undigested muscle fibers were present in large quantity. Brugsch and König found deficient absorption of fat, although the stools were normal in appearance.

Emaciation associated with suppurative pancreatitis is referred by some writers to functional disturbance of the gland, but is, in part at least, referable to continued suppuration. The accompanying weakness is often extreme.

Sequelæ.—Peritonitis due to extension of the inflammatory process to the surface of the organ and subsequent infection of adjacent serous surfaces is almost constantly associated with suppurative pancreatitis. Adhesions binding the posterior wall of the stomach to the pancreas may be the site of fistulous communications with this organ. Encysted peritonitis with effusion limited to the lesser peritoneal cavity is present in cases which pursue a subacute course. General peritonitis has been present in a considerable number of cases examined at autopsy. Rupture of the pancreatic or peripancreatic abscess may occur into a variety of organs. Perforation of the stomach or of the duodenum is not infrequent. Vomiting of purulent fluid and hemorrhage from the bowel have occasionally been associated with perforation of the duodenum. Rupture of an abscess into the general peritoneal cavity has been observed in 4 cases cited by Körte.

Thrombosis of the splenic vein extending to the portal vein may occur. In a case described by Bamberger the portal and splenic veins were almost completely occluded by a thrombus which had undergone puriform softening in the neighborhood of an abscess in the head of the pancreas; the liver was enlarged and contained numerous abscesses, the largest of which was the size of a hen's egg. Abscesses in the left lobe of the liver were present in cases of Frison and of Drasche.

Diagnosis.—Since suppurative pancreatitis in a considerable number of instances follows hemorrhagic inflammation, the symptoms of onset are those of the last-named condition, and indeed, even at operation, the distinction between abscess of the pancreas and gangrenous inflammation with peripancreatic abscess is often difficult, although fortunately not of much clinical significance. It is not improbable, however, that pancreatitis which is primarily suppurative may begin with equal severity. In another group of cases the onset is gradual, and there are vague symptoms of gastro-intestinal disturbance with epigastric pain of variable intensity; irregular fever, perhaps with chills, may suggest suppuration. When a deep-seated mass in the region of the pancreas appears, the diagnosis of peripancreatic abscess can be made, but it is rarely if ever possible to decide if peripancreatic abscess accompanies gangrenous inflammation or is the result of an abscess within the substance of the gland. Hence the differential diagnosis between hemor-

rhagic and suppurative pancreatitis is rarely possible, since, especially in the early stages of the disease, the symptoms are those of acute hemorrhagic pancreatitis, although the early onset of fever with chills and irregular temperature may suggest primarily suppurative pancreatitis. The symptoms which have been mentioned appearing in association with recognizable cyst, lithiasis, or neoplasm of the pancreas may suggest that suppuration has occurred as a complication of one of these lesions.

When a tumor-like resistance can be felt in the epigastrium behind the stomach and colon, its ill-defined character and its rapid appearance in association with acute symptoms serve to distinguish it from cyst or carcinoma. Nevertheless, the difficulty of diagnosis, especially when the abscess is small, is well illustrated by a case cited by Thayer. An abscess situated at the junction of the head and body of the gland caused complete obstruction of the common bile duct and a clinical picture suggesting carcinoma of the head of the pancreas or of the common bile duct.

Treatment.—Efficient treatment of abscess of the pancreas can be obtained only by surgical methods. Diagnosis is often impossible, but symptoms which indicate the occurrence of suppuration in or about the pancreas suggest the advisability of an exploratory laparotomy, and delay may be followed by grave complications, such as general peritonitis, thrombosis of the portal vein, and empyema. Nevertheless, the success of surgical treatment is limited by the character of the lesion; with diffuse purulent infiltration and with multiple abscesses of the gland, surgical procedures, according to Körte, will probably be unsuccessful; but when a large accumulation of pus is present within or about the gland, incision of the abscess, removal of its contents, and drainage of the cavity offer opportunity for recovery. Suppuration within the substance of the gland is in most instances rapidly followed by peripancreatic suppuration, but the surgeon who has evacuated pus contained within the bursa omentalis may be unable to examine the gland with thoroughness. In some of the cases upon which operation has been performed an abscess cavity has been limited to the substance of the pancreas.

CHRONIC INTERSTITIAL PANCREATITIS.

This is characterized by increase of interstitial tissue which partially replaces the parenchymatous cells of the organ, and is doubtless the most common disease to which the pancreas is subject. It is, however, accompanied by such ill-defined symptoms that it is rarely recognized during life unless an abdominal operation reveals its existence. When the lesion is associated with advanced destruction of the secreting parenchyma, digestive disturbances due to loss of pancreatic juice may result; in some cases the lesion is accompanied by diabetes mellitus. When the effects of chronic pancreatitis are compared with those of hepatic cirrhosis, it is not surprising that the ill-defined functional disturbances which result are not proportioned to the physiological importance of the gland. The symptoms of hepatic cirrhosis are not due to alterations of the metabolic and digestive functions of the liver, but are dependent upon the peculiar character of its circulation and to the facility with which the size of the organ may be determined. Such symptoms are not available for diagnosis of pancreatic disease.

Etiology.—The incidence is well illustrated by a series of 30 cases examined in the Johns Hopkins Hospital, 17 being in males and 13 in females. The ages of the individuals affected were as follows:

	Cases.
10 to 20 years	1
20 to 30 years	2
30 to 40 years	2
40 to 50 years	9
50 to 60 years	11
60 to 70 years	3
70 to 80 years	2

In five-sixths of the cases death occurred after the fortieth year, while more than two-thirds of the number occurred during the fifth and sixth decades of life. The relation of the disease to gallstones, carcinoma, and cirrhosis of the liver explains its occurrence during and after middle life.

Partial and complete occlusion of the duct of Wirsung, brought about by pancreatic calculi within it, by large gallstones lodged in the diverticulum of Vater, or by carcinoma or other new-growth, is the most frequent cause of chronic interstitial pancreatitis. The two ducts of the pancreas anastomose within the gland in nine of ten individuals, and occasionally the duct of Santorini is of large size, being in one of ten cases as large or larger than the duct of Wirsung, but in a much greater number of subjects it is rudimentary in some part; the duodenal orifice especially is often very minute and cannot act as an outlet for the entire pancreatic secretion.

Ligation of the pancreatic ducts in animals is followed by chronic inflammation. The irritant action of the retained secretions and in many cases bacterial invasion of the obstructed duct combine to produce sclerosis, but in human cases the relative importance of the two factors is difficult to determine. Pancreatic calculi, for example, are doubtless in many instances at least, produced by an infectious process within the ducts of the gland, and the associated sclerosis may be of such severity that the gland is converted into a fibrous band.

Much attention has been directed to the occurrence of chronic interstitial pancreatitis with cholelithiasis; the consequent induration of the gland when observed at operation undertaken for the removal of gallstones has not infrequently been mistaken for carcinoma affecting the head of the pancreas. Riedel, who first described this condition, found in 3 of 122 operations upon the bile passages such dense induration of the head of the gland that malignant growth was suspected; 2 of these patients recovered, while autopsy in a third demonstrated the presence of chronic interstitial pancreatitis. Robson and Moynihan have described 7 cases in which jaundice and other symptoms have suggested biliary colic, for the relief of which operation has been undertaken; the indurated condition of the head of the pancreas made a diagnosis of malignant disease not improbable, but the subsequent histories excluded this possibility. A small calculus lodged at the duodenal orifice of the diverticulum of Vater might leave unobstructed the orifices of the two ducts which enter the common channel, thus causing acute hemorrhagic pancreatitis by diverting bile into the pancreatic duct, but a large stone in the same position would occlude the pancreatic duct and cause chronic interstitial inflammation similar to that which follows ligation of the duct in animals.

There is little doubt that chronic pancreatitis may follow cholelithiasis, even though no occlusion of the pancreatic duct has been caused by calculi;

in such cases acute inflammation of the ducts of the pancreas accompanies similar inflammatory changes in the biliary passages consequent upon the presence of gallstones in the gall-bladder. There is abundant evidence that ascending infection of the pancreatic duct, either from the inflamed bile passages or from the duodenum, may be a cause of chronic pancreatitis. Both Körte and Flexner have caused chronic pancreatitis by injecting colon bacilli and other microorganisms into the pancreatic duct of animals.

In cases of advanced chronic interstitial inflammation examined at autopsy, persistent vomiting accompanied by nausea and epigastric pain had afforded evidence during life of gastric or gastro-intestinal disease. It is not improbable that the persistent vomiting present in these cases produced conditions favoring ascending infection of the pancreatic ducts.

The causes of chronic pancreatitis just considered are such as affect the gland by way of its ducts. In a smaller group of cases the lesion is referable to the bloodvessels or to toxic substances carried to the gland by them. Arteriosclerosis causes chronic pancreatitis, according to Hoppe-Seyler and Fleiner, who have described such cases, and the lesion is analogous to the contracted kidney and to the changes in the liver, heart, and brain following arterial disease. In a case of diabetes with calcification of the posterior tibial artery and gangrene of the foot, chronic pancreatitis, included among the 30 cases previously cited, accompanied advanced sclerosis of the pancreatic vessels; Hoppe-Seyler has recently described 9 similar cases. By some writers, notably Hansemann and his pupil Kasahara, syphilis has been thought to be the most frequent cause of diffuse interstitial inflammation of the gland.

Since alcohol is regarded as one of the most important factors in the production of cirrhosis of the liver, its relation to the similar lesion of the pancreas has much interest. The literature of pancreatic disease contains numerous instances recorded by Friedreich, Chvostek, Dieckhoff, Oser and others in which the lesion has been found in individuals addicted to excessive use of alcohol, and especially noteworthy is the fact that the lesion is in these cases frequently, although not necessarily, associated with cirrhosis of the liver.

Factors other than alcohol producing chronic inflammation act simultaneously upon the liver and pancreas, and chronic pancreatitis and cirrhosis have been found associated by many observers, notably by Kasahara, by Lefas, and by Steinhaus¹; in 6 cases of cirrhosis the former found marked increase of interstitial tissue in the pancreas in 2, and moderate increase in 2. Among 30 cases of chronic pancreatitis, in 8 cirrhosis was present. It is improbable that changes in the portal circulation caused by cirrhosis of the liver produce the very marked increase of connective tissue found in the pancreas, since chronic passive congestion due to other causes is rarely accompanied by the lesion. An example of the dependence of chronic inflammation of the liver and pancreas upon the same condition is furnished by the disease known as hemochromatosis, in which an iron-containing pigment is deposited in the cells of the liver, pancreas, and various other organs. Cirrhosis of the liver with hypertrophy results, while chronic pancreatitis is accompanied by diabetes mellitus, which is the usual cause of death in these cases.

¹ *Deutsch. Arch. f. klin. Med.* 1902, lxxiv, 537.

Special Pathology.—Interest in this has in large part limited itself to the relation of the lesion to diabetes mellitus, and since chronic interstitial pancreatitis may occur with or without diabetes attempts have been made to define the peculiarities of those lesions which cause a disturbance of carbohydrate metabolism. According to the distribution of the newly formed connective tissue, so-called interlobular, peri-acinous, and monocellular types have been described, while in some instances proliferation of interstitial tissue has been thought to have a peculiar relation to the bloodvessels. Hansemann has described a form of chronic pancreatitis which is, he believes, peculiar to diabetes. He designates the condition atrophy, but carefully distinguishes it from simple atrophy due to cachexia, and defines it as a type of chronic inflammation resembling certain forms of granular atrophy of the kidney. The organ is diminished in size, and is therefore distinguishable from what he designates fibrous induration with hypertrophy, which in three cases he found associated with diabetes. In a recent paper Hansemann has defined with greater detail the microscopic characters of so-called granular atrophy of the pancreas, and states that it agrees substantially with what has been subsequently called interacinar pancreatitis; he does not find, however, the constant implication of the islands of Langerhans, to which has been attributed disturbance of carbohydrate metabolism.

Two types of chronic interstitial pancreatitis are distinguishable. In one type broad bands of newly formed connective tissue occupy the site of the larger trabeculae of the gland and separate the lobules or groups of lobules; areas of parenchyma thus surrounded may be wholly uninvaded by the sclerotic process; the lesion may be designated chronic interlobular pancreatitis. With the second type of chronic inflammation newly formed connective tissue does not form broad bands, but occurs diffusely scattered in small patches and strands, which invade the lobules of parenchyma and separate individual acini. The lesion may be called chronic interacinar pancreatitis. Among 30 instances of chronic pancreatitis, in 21 the lesion was of the interlobular type and in 9 of the interacinar type.

Chronic interlobular pancreatitis results when the duct of the gland is occluded or when the inflammatory process is the result of ascending infection of the ducts. Typical interlobular pancreatitis can be produced in cats by ligation of the ducts of the gland; at the end of a week or more each lobule which in this animal is unusually sharply defined, is separated from its neighbors by thickened bands of connective tissue. In human cases the most intense grades of pancreatic sclerosis occur in association with duct obstruction resulting from the presence of pancreatic calculi. The organ may be converted into a narrow band of dense scar-like tissue surrounding the moderately dilated duct filled with numerous concretions. To the naked eye the tissue of the organ altered by interlobular inflammation is firmer than usual, and on section is compact in texture, the areolar character of the interstitial tissue being lost.

At an early stage of the lesion the newly formed tissue shows little tendency to invade the lobules of the parenchyma, and in most instances only their peripheral parts contain invading strands of tissue. At a later stage entire lobules and groups of lobules are completely destroyed, so that a group of several widely separated and atrophied acini embedded in cellular interstitial tissue may be all that remains of one or more lobules. Characteristic both of the experimental and of the human lesion which follows occlusion of

the pancreatic ducts is the behavior of the islands of Langerhans. Since the cells forming the islands of Langerhans have no communication with the ducts of the pancreas and take no part in the elaboration of the pancreatic juice, it is not surprising that they are less affected than the cells of the secreting acini, and often remain wholly uninjured, although the secreting parenchyma has almost completely disappeared. Since intervening acini are replaced by scar-like connective tissue, islands of Langerhans which have been previously separated are brought close together, so that in a given area their number may appear to have greatly increased. Embedded in dense, fibrous tissue, which in time undergoes contraction, the resisting islands of Langerhans finally suffer, possibly as the result of interference with their blood supply. The cells of these compressed structures atrophy, and small groups of much atrophied cells scattered in dense stroma may be all that remains of these bodies.

With *chronic interacinar pancreatitis*, characterized by a diffuse new-growth of interstitial tissue penetrating between the acini, the organ is tough rather than hard, and does not exhibit the nodular surface seen with the interlobular type of inflammation. In some instances the organ shows no noteworthy macroscopic change and the lesion is demonstrable only by microscopic examination. The newly formed interstitial tissue is somewhat irregular in distribution, so that at one point there may be diffuse thickening of the strands of connective tissue between the acini, while elsewhere occur larger bands or masses of stroma. The interlobular tissue is not unaffected, but its proliferation is not a constant feature of the lesion, so that the lobulation of the organ is not accentuated, but rather obscured, since irregularly scattered strands of new tissue tend to obscure the interlobular boundaries.

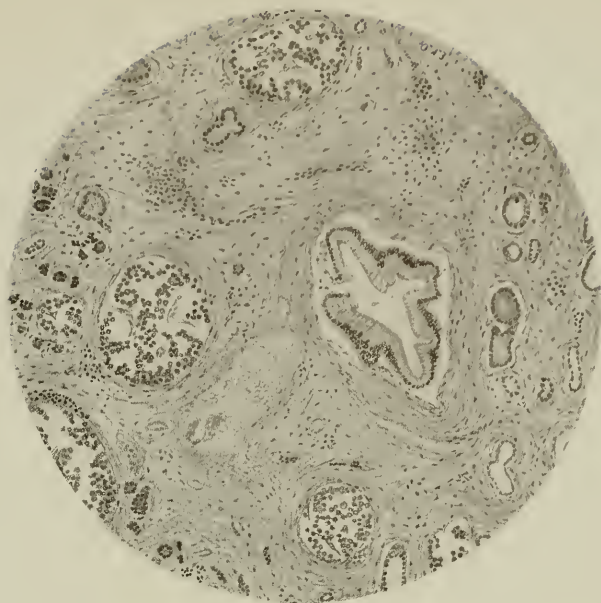
With the interlobular type of inflammation the islands of Langerhans persist, and are affected only when the lesion has reached a very advanced stage; but with the interacinar type, on the contrary, these islands are implicated even when the lesion is little advanced. They are surrounded by fibrous tissue, which forms a capsule separating them from adjacent acini. Within the islands there may be proliferation of interstitial tissue forming coarse strands, which follow the course of the capillaries and separate the columns of epithelial cells. With increase of stroma the cells atrophy and disappear, so that the entire structure is replaced by a minute fibrous scar.

Chronic interlobular pancreatitis, in many instances at least, is referable to an irritant which attacks the glands by way of its ducts. In those instances in which the etiology of the interacinar type is evident the exciting cause, on the contrary, seems to reach the organ by way of the bloodvessels. Where chronic inflammation is a consequence of arteriosclerosis, it is of the interacinar type; observations of Lefas and of the writer show that many cases of chronic pancreatitis, associated with atrophic cirrhosis of the liver, and presumably due to the action of the same cause, which is in many instances alcohol, are interacinar in type. In some cases, however, the etiology of interacinar pancreatitis is obscure.

Chronic interstitial inflammation of the pancreas, which accompanies the disease known as *hæmochromatosis* (or when accompanied by diabetes mellitus as bronzed diabetes), affects the interacinar tissue and implicates the islands of Langerhans; there may be in addition some thickening of the strands of connective tissue between the lobules of the gland. The deposi-

PLATE X

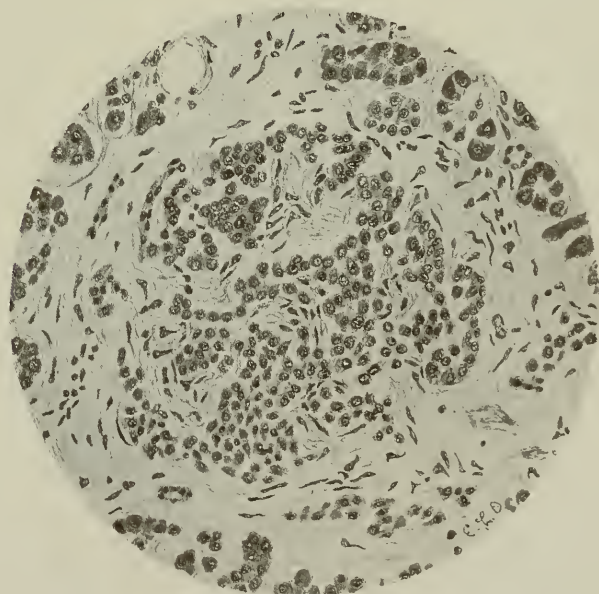
FIG. 1



Chronic Interstitial Pancreatitis Following Obstruction of Pancreatic Duct.

Showing unchanged islands of Langerhans embedded in sclerotic tissue.

FIG. 2



Chronic Interacinar Pancreatitis with Diabetes Mellitus.

Showing invasion of the islands of Langerhans by fibrous tissue.

(From Journal of Experimental Medicine.)

tion of an iron-containing pigment in the liver, pancreas, and other glands is accompanied by degeneration of the parenchymatous cells and chronic interstitial inflammation of these organs. Bronzing of the skin due to deposition of pigment occurs, and in the later stages of the disease there is diabetes mellitus, which is the usual cause of death. The pancreas is firm in consistence and of a chocolate-brown color due to the presence of yellowish-brown pigment both in the parenchymatous cells and in the interstitial tissue of the organ. In a case of hæmochromatosis examined, included among the 30 instances of chronic pancreatitis before mentioned, the interstitial tissue of the gland was much increased, diffusely distributed, and separated individual acini or groups of acini. Islands of Langerhans were abundant throughout the gland, but were surrounded by a thickened capsule of fibrous tissue, and were occasionally invaded by thickened strands. In an instance of the same disturbance of pigment metabolism described by Beattie,¹ accompanied by diabetes (bronzed diabetes), there was increase of the interstitial tissue both between and within the lobules; the islands of Langerhans were scanty in number, perhaps replaced by small scars which were present; those islands which persisted were surrounded by dense capsules of fibrous tissue.

Clinical Significance of Chronic Interstitial Pancreatitis.—Chronic pancreatitis is rarely accompanied by such definite symptoms that its diagnosis is possible, but the relation of the disease to other recognizable lesions and its association with diabetes mellitus may suggest its probable existence. Symptoms such as severe pain in the epigastrium and in the midscapular region, vomiting, and loss of weight and strength have been noted in association with chronic inflammation of the gland, but are not referable with certainty to the disease which frequently accompanies grave disturbances of the stomach, duodenum, or bile passages. Disturbance of digestion due to loss of pancreatic juice and indicated by the presence of fatty stools may occur in association with chronic pancreatitis, but is usually the result of carcinoma, cysts, or calculi, which occlude the duct, and as a consequence cause chronic inflammation.

Probable existence of chronic pancreatitis may be suggested by lesions of adjacent organs, particularly when such lesions are accompanied by glycosuria. Chronic interlobular pancreatitis is almost constantly present in association with carcinoma of the head of the pancreas and with pancreatic calculi, but only when the lesion is advanced and partial destruction of the islands of Langerhans has occurred does diabetes mellitus ensue. Hence glycosuria is present in a relatively small proportion of instances of pancreatic lithiasis and of carcinoma of the gland, and rarely, if ever, accompanies chronic pancreatitis caused by biliary calculi. Since glycosuria in association with carcinoma, cyst, or calculi indicates far advanced chronic disease of the pancreas, it has grave prognostic significance.

Riedel and Mayo Robson have emphasized the importance of recognizing at operation chronic interstitial pancreatitis occurring with cholelithiasis. In a certain number of operations undertaken for the relief of gallstones the head of the pancreas is so indurated that the existence of carcinoma is suspected. In several cases a grave prognosis has been based upon the discovery of such induration, but the recovery of the patient has proved

¹ *Journal of Pathology and Bacteriology*, 1903, ix, 117.

the absence of malignant growth. Mayo Robson believes that chronic pancreatitis should be regarded as a serious complication of gallstones, at times causing death, with increasing weakness and emaciation; cholecystotomy with drainage of the gall-bladder and removal of the calculi, when necessary, is, he maintains, followed by recovery.

While chronic interlobular pancreatitis is accompanied by diabetes mellitus in only a small proportion of cases, and occurs only when the lesion is far advanced, chronic interacinar pancreatitis which implicates the islands of Langerhans is in almost all cases accompanied by glycosuria. The disease causes relatively slight destruction of the secreting parenchyma and is rarely accompanied by symptoms, other than glycosuria, referable to the gland. Lancereaux believed that diabetes with emaciation was referable to a lesion of the pancreas, whereas diabetes with obesity was unaccompanied by lesion of the organ; but few observers have accepted these criteria. In a large proportion of cases it will be impossible to decide during life if diabetes is referable to interacinar pancreatitis. When, however, diabetes mellitus occurs in an individual suffering from cirrhosis of the liver, the former disease is with considerable probability dependent upon the co-existence of chronic interacinar pancreatitis, doubtless produced by the same etiological factor, in some cases alcoholic excess, which is responsible for the hepatic disease. In another group of cases diabetes mellitus accompanied by advanced arteriosclerosis may with much probability be referred to chronic interacinar pancreatitis caused by the arterial disease.

TUBERCULOSIS OF THE PANCREAS.

Miliary tuberculosis of the pancreas in association with acute or chronic tuberculosis of other organs is not uncommon, but widespread tuberculosis causing functional disturbance or recognizable enlargement of the gland is almost unknown. Among 128 cases of tuberculosis Kudrewetsky found tubercles in the pancreas in nearly a tenth of the cases; they were common with acute miliary tuberculosis and with tuberculosis in children. Large tuberculous lesions are uncommon; Chvostek described a case in which the enlarged pancreas formed a fibrous mass containing caseous areas the size of a walnut. In a case described by Sandler a small tumor was felt above the umbilicus in a woman who had suffered with pain and vomiting during nine months; a small mass removed at operation from the head of the pancreas was found to be a tuberculous lymphatic gland. Carnot mentions a case in which the splenic end of the pancreas in contact with a tuberculous kidney was the seat of tuberculosis.

Chronic pancreatitis has been found by Carnot associated with tuberculous lesions of other organs; in 7 cases of tuberculosis he found a moderately advanced increase of connective tissue about the bloodvessels and between the lobules of the gland.

SYPHILIS OF THE PANCREAS.

Congenital syphilitic pancreatitis was found by Birch-Hirschfeld in 29 of 124 syphilitic newborn infants. With this lesion increase of the inter-

stitial tissue prevents the development of the secreting parenchyma; the islands of Langerhans resist the process better than the secreting parenchyma and persist, surrounded by connective tissue. Miliary gummata occur occasionally within the connective tissue which has undergone proliferation.

The pancreas is rarely the seat of gummata occurring as the result of acquired syphilis. From the older literature Friedreich cited three cases in which gummata of the pancreas were associated with characteristic syphilitic lesions of other organs. Cases of pancreatic syphilis in which the lesion is analogous to that of syphilis of the liver have been described by Drozda and by Schlagenhauser; in association with syphilitic lesions in other parts of the body the pancreas has been found to contain scar-like masses of connective tissue within which are caseous gummata, in one instance the size of a hazelnut. In a case of Chvostek the tail of the pancreas was penetrated by sclerotic bands which gave it a lobed appearance; gummata were not present. There are no data with reference to the symptoms or diagnosis of syphilis of the pancreas.

By some writers syphilis has been believed to cause chronic interstitial inflammation affecting diffusely the entire gland. In two cases of chronic pancreatitis associated with diabetes mellitus, Hansemann obtained evidence of syphilis. In 2 of 6 cases of acquired syphilis Kasahara found a moderate increase of interstitial tissue. Syphilis is not, as these writers believe, the most frequent cause of chronic pancreatitis, for in only 1 of 30 cases of chronic inflammation of the pancreas examined in the Johns Hopkins Hospital was there a history of syphilis, and in this case chronic pancreatitis was the result of carcinoma compressing the duct of Wirsung.

CALCULI OF THE PANCREAS.

Pancreatic calculi occur much less frequently than gallstones, which in most instances are formed within the gall-bladder, and not like pancreatic stones within ducts. The frequency of the disease is doubtless much exaggerated by the statistics of Giudiceandrea, who found pancreatic calculi twice among 122 autopsies. Two instances occurred among 1500 autopsies at the Johns Hopkins Hospital.

Unlike gallstones, pancreatic calculi occur more frequently in men than in women; among 57 cases collected from the literature by Lazarus,¹ 47 were in men and 10 in women, a ratio of about 5 to 1. In almost two-thirds of these cases the disease affected individuals between the ages of thirty and fifty years.

Etiology.—Observations and experiments have shown that cholelithiasis is usually associated with bacterial infection of the gall-bladder or bile passages, and in many instances it has been possible to demonstrate bacteria, not infrequently forming masses within the calculus. The mucous membrane of the gall-bladder is usually the site of an inflammatory process accompanied by desquamation of epithelium and increased production of mucus, together with other changes in the composition of the bile which are not clearly understood. Obstruction to the outflow of bile from the gall-

¹ *Beiträge zur Pathologie und Therapie der Pankreaserkrankungen*, Berlin, 1904.

bladder is essential to the experimental production of biliary calculi; otherwise, bacteria which have been injected fail to gain a foothold and are expelled with the bile. Many analogies suggest that similar factors are concerned in the production of pancreatic calculi.

The presence of calculi is usually associated with acute and chronic inflammatory changes in the ducts and in the parenchyma of the gland, but etiological relation of these factors to lithiasis is doubtful, since, in part at least, they are caused by occlusion of the ducts or by secondary invasion of bacteria. Giudiceandrea was able to demonstrate the colon bacillus together with numerous long bacilli within pancreatic calculi, while in a second case he found in the centre of a calculus numerous cocci and bacilli differing much in morphology.

The association of pancreatic and biliary calculi in a considerable number of cases cited by Ancelet (eight cases), and observed by Curnow, Dieckhoff, and Lazarus, suggests the possibility that ascending affection from the bile passages, favored perhaps by temporary obstruction of the pancreatic ducts by passing gallstones, may have caused pancreatic lithiasis, although perhaps both biliary and pancreatic calculi may have been due to the same cause. That stagnation of pancreatic secretion favors the formation of calculi is indicated by cases in which calculi, at times in considerable number, are found within pancreatic cysts. Occasionally an impacted stone has caused the formation of a retention cyst, but in other cases large cysts of obscure origin have contained numerous calculi; Lazarus has observed 4 such cases.

Attempts have been made to produce pancreatic calculi, and in a few instances very small concretions have been formed experimentally. Thiroloix injected sterilized soot into the pancreatic duct, and found small concretions in a cyst in the tail of the gland and in the pancreatic duct. Lazarus found a minute concretion in a cyst experimentally produced by local degeneration and softening of parenchyma. Desquamated epithelial cells clumped together formed the organic nucleus of the concretion.

Special Pathology.—In some cases the pancreatic ducts contain fine sand-like concretions, but more frequently several stones, perhaps a centimeter or more in diameter, are present. The largest recorded calculus (Schuppmann) was stellate in shape and measured one and a half inches in its largest diameter. A solitary calculus is rarely found, while several hundred varying greatly in size may be present. In some instances projections from a large stone situated in the large duct extend into adjacent branches, so that a cast of the dilated ducts is produced. In consistence pancreatic concretions vary considerably, being at times mortar-like and friable, but usually hard; they may have a nucleus of organic material consisting of cell detritus. Pancreatic calculi are white, often grayish or yellowish white, but in rare instances black or brownish-black concretions have been found within the ducts.

Biliary calculi have in several cases been found within the duct of Wirsung near its junction with the common bile duct. In an example of suppurative pancreatitis, described by Dieckhoff, the duct of Wirsung was dilated and contained a gallstone.

Pancreatic calculi are composed in great part of inorganic salts, of which calcium carbonate and calcium phosphate are most abundant, although sodium phosphate, magnesium phosphate, and other salts may be present. Calculi consisting almost wholly of calcium carbonate or calcium phosphate

have been described. The composition of calculi from two cases described by Johnston¹ was as follows: (1) Calcium phosphate, 72.3 per cent.; calcium carbonate, 18.9 per cent.; organic matter, 8.8 per cent. (2) Calcium carbonate, 91.65 per cent.; magnesium carbonate, 4.15 per cent.; organic matter, 3 per cent.

In most instances inorganic constituents are more abundant than organic material, but in a case described by Baldoni organic material formed the greater part of the concretion.

A calculus having a nucleus of calcium carbonate surrounded by a layer of cholesterin has been described by Freyhan. It is not improbable that cholesterin is derived from degenerate or desquamated epithelial cells of the pancreatic ducts, since it is known to occur in columnar epithelial cells. A calculus consisting of calcium oxalate has been found at operation in a pancreatic cyst by Shattuck.²

Calculi may be situated in the larger ducts or their branches, but calculi of considerable size are usually found in the duct of Wirsung, of which the lumen is frequently so completely occluded that outflow of secretion is prevented and dilatation behind the obstruction results. Stones embedded in ampulla-like diverticula of the large duct have been described. Since the duct of Wirsung is narrower near its duodenal orifice than elsewhere, calculi tend to lodge in this situation, and the duct may be uniformly dilated to a diameter of a centimeter or more. Stones lodged in the terminal part of the duct of Wirsung may compress the common bile duct and cause jaundice; Gould found in the diverticulum of Vater a pancreatic calculus which had caused jaundice.

Obstruction to the outflow of pancreatic juice, doubtless in association with bacterial infection, causes chronic interstitial inflammation of the pancreas, which may reach such an advanced stage that the gland is converted into dense fibrous tissue surrounding the dilated duct, secreting parenchyma having almost completely disappeared. The cells of the acini atrophy and are replaced by new connective tissue, coarse bands separating those lobules of parenchyma which are still intact. This interlobular type of sclerosis tends to spare the islands of Langerhans, which long remain almost unchanged.

The most intense sclerosis to which the gland is subject occurs in association with calculi. In the contracting fibrous tissue, the islands of Langerhans finally undergo changes, being compressed and invaded by the sclerotic tissue. In cases of diabetes with pancreatic lithiasis such isolated islands of Langerhans had suffered such changes (Lazarus, Pearce, Lancereaux, etc.).

Calculi situated within the ducts not infrequently cause ulceration of the mucosa by pressure. In the immediate neighborhood of a stone chronic inflammation more intense than elsewhere may produce abundant scar tissue, distorting the duct. The presence of bacteria, the development of which is favored by obstruction of the ducts, may result in the formation of pancreatic *abscess*, which is not an infrequent result of pancreatic calculi.

Symptoms.—Pancreatic lithiasis may be unaccompanied by noteworthy local symptoms indicating disease of the organ. Lazarus de-

¹ *American Journal of the Medical Sciences*, 1883, lxxvi, p. 404.

² *British Medical Journal*, 1896, i, 1034.

scribed such a case in which death occurred with diabetes and pulmonary tuberculosis. At autopsy the pancreas was found to be converted into a fibrous sac surrounding the much dilated duct filled with concretions of varying size; neither on the surface of the organ nor on section was glandular tissue to be seen. Yet, notwithstanding the fact that the duct contained numerous jagged concretions, there had been no pain.

The most frequent symptom is *pain*, which may be continued or intermittent; in some instances there is merely a sense of pressure. More characteristic are attacks of colicky pain, which in some instances have been regarded as sufficiently distinctive for diagnosis of the disease. Friedrich doubted the occurrence of *pancreatic colic* analogous to gallstone colic. Subsequent observations have shown that severe attacks of colic-like pain occur in association with the passage of calculi. In a case of Leichtenstern's, symptoms of biliary colic without jaundice were followed by the passage with the feces of three concretions, the largest the size of a pea; analysis showed that they were composed of calcium carbonate without traces of either bile pigments or cholesterin. Pain has been localized in the epigastrium and under the left costal margin in the mammillary line, radiating on the left side to the spine or to the left shoulder-blade. Pain with pancreatic lithiasis does not necessarily occur to the left of the mid-line, but may be limited to the region of the epigastrium or of the umbilicus, or may even radiate to the right side (Eichhorst).

As with biliary colic, the attacks of pain associated with passage of pancreatic calculi are often accompanied by vomiting. Reflex circulatory disturbances, such as weak pulse and temporary collapse, may occur. The pain is at times accompanied by a chill which is followed by fever, possibly referable to bacterial invasion of the occluded duct; with the subsidence of the attack the temperature falls.

The passage of a pancreatic calculus through the diverticulum of Vater may cause temporary *jaundice*. A patient of Leichtenstern had suffered during two years with attacks of a severe abdominal pain accompanied by vomiting and on one occasion by jaundice, but cholecystotomy undertaken for the relief of gallstones demonstrated their absence; autopsy showed the presence of numerous pancreatic calculi. The relatively frequent association of cholelithiasis with pancreatic lithiasis may explain the occurrence of jaundice in some instances.

In several instances a diagnosis of pancreatic lithiasis has been made during life from the passage with the feces of calculi composed of calcium carbonate or phosphate and free from bile pigments or other constituents of the bile. In a case of Minnich during twelve days following an attack of colic, soft friable concretions as large as a cherry pip were found, containing carbonate and phosphate of calcium. In a case of Caparelli over one hundred small pancreatic calculi were passed from the fistulous opening of an abscess in the epigastric region.

Diabetes mellitus, or alimentary glycosuria, has been a symptom of pancreatic lithiasis in a large proportion of the recorded cases; Lazarus has collected 80 cases, among which glycosuria occurred in 36 (45 per cent.). Pancreatic lithiasis is an infrequent cause of diabetes, occurring only 5 times among 288 cases of diabetes recently collected from the literature. Pancreatic lithiasis may exist for years without diabetes, and in the case described by Caparelli glycosuria appeared six years after the first attack

of colic. Since glycosuria is not the result of occlusion of the pancreatic ducts, but of consequent chronic interlobular inflammation, diabetes results only after the lesion has become far advanced and has injured the islands of Langerhans. At one stage of the disease the organ is subject to such slight impairment of function that *alimentary glycosuria* is present; in cases of Lichtheim, Lazarus, and Opie, glycosuria disappeared when the patient was given a diet poor in carbohydrates. In cases of Polyakoff and of Minnich transient glycosuria occurred only after attacks of colic, and was doubtless analogous to that which not infrequently follows experimental injury to the gland, insufficient to cause permanent diabetes.

Steatorrhœa has been present in 10 of the cases collected by Lazarus. In the table of Fitz, which contains 29 cases in which visible fatty stools have accompanied demonstrable pancreatic disease, pancreatic lithiasis was present in 7 instances. In none of the cases of Fitz was jaundice present, while in 4 there was glycosuria. In cases studied by Müller and by Kinnicutt and Herter, although fat was not visible in the fœces, chemical analysis demonstrated that the percentage of split fat was much less than normal.

Azotorrhœa, shown by the presence of undigested muscle fibres in the fœces, is even less common than fatty stools, and in only one (Lichtheim) of the eight instances in which this symptom accompanied well-authenticated pancreatic disease (Fitz) was the pancreatic lesion caused by calculi.

The *loss of weight* which occurs in association with pancreatic lithiasis in the absence of diabetes or disturbances of digestion is probably referable, in part at least, to inflammatory changes or other complication of the disease.

Complications.—Among these the most frequent and serious is *abscess*. Rupture may occur into the peritoneal cavity causing a localized bursal abscess or fatal general peritonitis. A fistulous communication with the intestinal tract may be formed; in a case described by Nicolas and Mollière one liter of blood was passed by rectum two and a half months after an attack of severe epigastric pain; a second attack of colic and subsequent hemorrhage, together with chills, fever, and glycosuria, preceded death. The duct of Wirsung contained about twenty concretions, while in the substance of the gland communicating by a fistulous opening with the duodenum was an abscess from which hemorrhage had occurred. In the case of Caparelli an abscess opened near the umbilicus, and calculi were passed from the fistula which persisted for six years.

The association of cysts with pancreatic calculi has already been mentioned. Cases in which *carcinoma* of the pancreas has been described in association with calculi date from a time when chronic inflammation was not infrequently mistaken for scirrhus carcinoma. The frequent association of carcinoma of the gall-bladder and gallstones has little analogy in the pancreas. In a case of Schupmann carcinoma occupied the tail of the gland, and in the region of the tumor within the duct was a large jagged concretion.

Diagnosis.—Kinnicutt has collected from the literature seven cases in which a diagnosis of pancreatic lithiasis has been made during life. In the case of Caparelli calculi were discharged from a fistula in the epigastric region, and in the cases of Leichtenstern, Minnich, and Kinnicutt calculi consisting of phosphate or carbonate of calcium without mixture of bile

salts were passed with the stools after attacks of epigastric colic. An additional case has been described by Cipriani.¹

A review of the recorded cases gives no data by which it is possible to distinguish pancreatic from biliary colic, although localization of the pain below the left costal margin may be suggestive of pancreatic lesion. Diabetes mellitus, or alimentary glycosuria, may indicate a lesion of the gland. Fatty stools have been less frequently observed, and in the absence of jaundice have diagnostic value; diminution of split fat in the feces is believed to have similar significance. Jaundice may be caused by a pancreatic calculus lodged in the diverticulum of Vater, or by biliary calculi, which occur with relative frequency in association with pancreatic calculi, and does not exclude a diagnosis of pancreatic colic. In the case of Lichtheim severe attacks of colic with fever and vomiting had been unaccompanied by jaundice, although they had been of long duration; the site of pain and the absence of urinary symptoms served to exclude renal calculi, while the occurrence of diabetes mellitus furnished evidence that the colic was referable to a pancreatic lesion.

Treatment.—Statements concerning this are necessarily vague, since few cases have been recognized during life. Attacks of colicky pain have been treated by sedatives, applicable to biliary colic. Measures which increase the flow of pancreatic juice have been suggested for the purpose of overcoming obstruction to its outflow, particularly since duct obstruction is believed to be an important factor in the production of calculi. Physiological observations according to Lazarus suggest that this may be accomplished by water in abundance, especially when carbonized or acidified by other means; alkalies are thought to diminish pancreatic secretion. To increase the flow of pancreatic juice, Eichhorst injected pilocarpine subcutaneously in a case believed to be pancreatic lithiasis, and found that attacks of colic disappeared. It is noteworthy in this connection that pilocarpine administered to animals with the pancreatic ducts ligated caused fat necrosis of almost the entire abdominal fat, and death within four days.

The presence of glycosuria indicates that the pancreas has undergone grave sclerotic change; nevertheless in several instances diabetic diet has caused the disappearance of sugar.

Analogy with gallstones suggests the possibility of surgical treatment. Körte regards as feasible the removal of stones from the duct of Wirsung, and cites a case in which he removed through the substance of the pancreas a gallstone lodged in the lower part of the common bile duct. Gould² found at operation upon a patient suffering with symptoms of cholelithiasis a lump at the posterior part of the lesser peritoneal cavity, and on incision removed a calculus from the duct of Wirsung. Obstruction to the flow of bile was not relieved, and at a second operation a stone was removed from the head of the pancreas; although bile subsequently entered the intestine, death occurred as the result of suppuration twelve days later. Moynihan³ removed through the duodenum a calculus lodged in the duct of Wirsung and projecting into the diverticulum of Vater.

¹ *American Journal of the Medical Sciences*, 1902, cxxiv, 948.

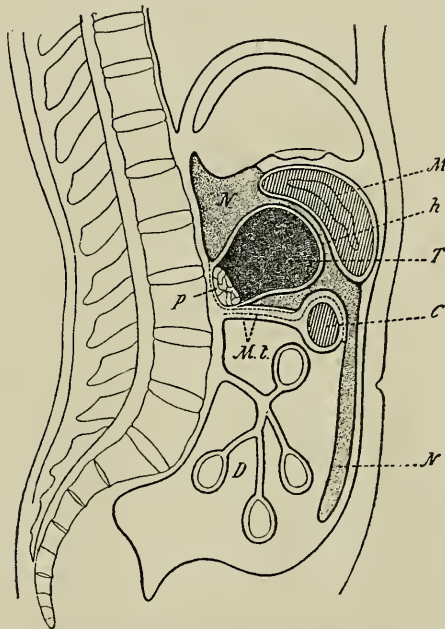
² *Lancet*, 1898, ii, 1632.

³ *Ibid.*, 1902, ii, 355.

CYST OF THE PANCREAS.

A variety of lesions are described as cysts of the pancreas; their common character is the presence in the upper part of the abdominal cavity of an encapsulated collection of fluid in contact with the gland and apparently derived wholly or in part from it. Since the greater number of such cysts are observed at operation, the exact nature of the disease is often obscure and its relation to the pancreas is not always established, particularly because fluid encapsulated by the thickened wall of the bursa omentalis may produce physical signs which are indistinguishable from those of pancreatic cysts situated within the substance of the diseased gland.

FIG. 8

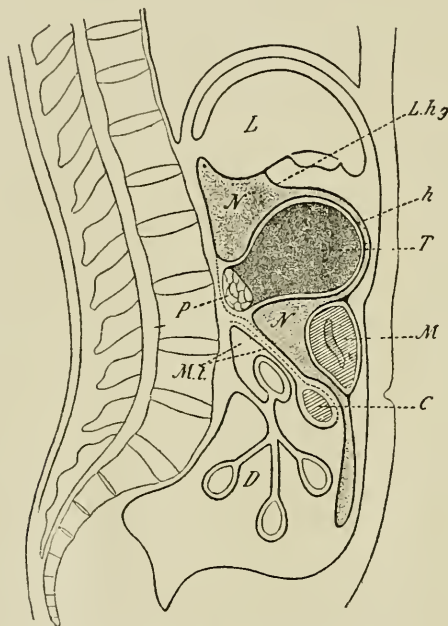


Cyst projecting into the lesser peritoneal cavity behind the stomach and above the colon.
(Oser.)

Classification.—True cysts derived from the ducts or acini of the pancreas and lined by a layer of epithelial cells have been distinguished from so-called pseudocysts, which have walls of thickened connective tissue and are without epithelial lining. True cysts may be formed by dilatation of the pancreatic duct or its branches due to obstruction, and are retention cysts, or may be the result of spontaneous proliferation of epithelial structures, and are tumors. Pseudocysts are the result of trauma or of degenerative changes within the substance of the gland, and frequently contain blood or blood mixed with products of pancreatic secretion; pseudocysts may be situated wholly within the substances of the pancreas, or, when associated

with rupture of the anterior surface of the gland, may be limited in part by the walls of the bursa omentalis. Pseudocysts, it has already been shown, may be the result of acute hemorrhagic pancreatitis. The etiology and course of these diverse lesions vary so greatly that it would be desirable to consider them separately were they not characterized by certain physical signs common to all encapsulated collections of fluid having their origin in the pancreas. The relation of different parts of the pancreas to adjacent structures determine the location of such cysts and the point at which they present upon the surface of the body. From an analysis of 133 cysts upon which operation had been performed Körte has described the various positions which they may occupy.

FIG. 9



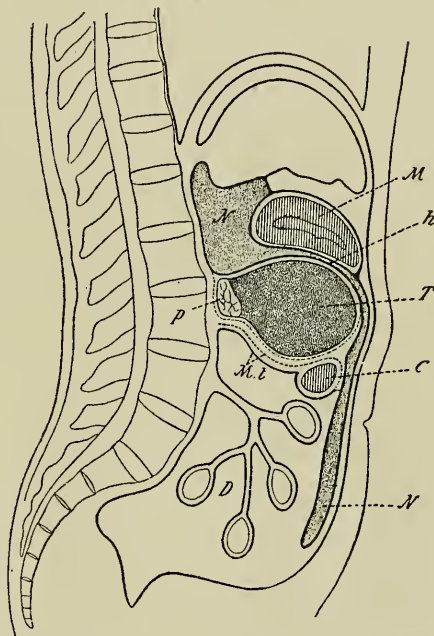
Cyst projecting into the lesser peritoneal cavity, and in contact with the abdominal wall between the stomach and pancreas. (Oser.)

A. In the majority the cyst grows directly forward, and, pushing the stomach upward, comes in contact with the anterior abdominal wall between the stomach and colon; in order to reach the tumor at operation it is necessary to cut through the gastrocolic omentum. The cyst occupies and distends the cavity of the bursa omentalis, and, stretching the gastrocolic ligament, may, with increased growth, push downward the transverse colon so that it may reach the symphysis pubis. Pseudocysts which arise from the pancreas and, as the result of rupture of its overlying peritoneum, communicate with the bursa omentalis, and are in large part limited by this cavity, occupy the position described and present upon the surface of the abdomen between stomach and colon. Jordan Lloyd has shown that accumulations of fluid

in the lesser peritoneal cavity due to a variety of causes may closely resemble these cysts of the pancreas. The stomach is compressed and flattened, but usually pushed upward. Should adhesions be formed between the cysts and the stomach, the latter may be held in front of the tumor and drawn downward by it.

B. Less frequently the cyst makes its way above the lesser curvature of the stomach between the stomach and liver, stretching the gastrohepatic omentum which covers it. The upper border of the body of the pancreas is at a higher level than the lesser curvature of the stomach, so that a cyst arising from this part of the gland may present above the stomach, which, with further growth of the tumor, is pressed downward; with gastropexia

FIG. 10



Cyst projecting from the upper surface of the mesocolon and presenting between stomach and colon. (Oser.)

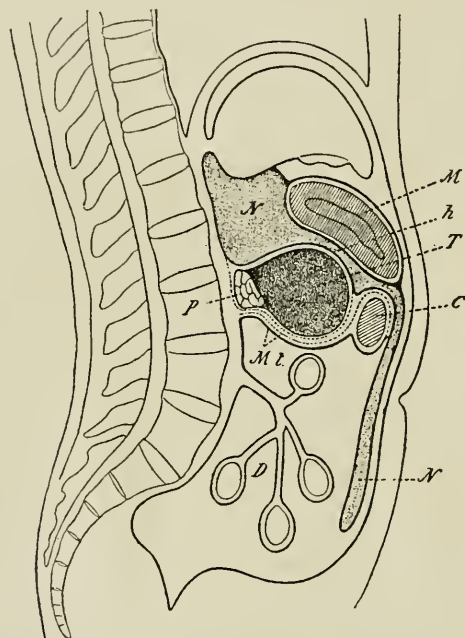
the extent of pancreas above the lesser curvature of the stomach is increased. Körte cites from the literature 9 cases in which the cyst presented at operation above the stomach, and in 2 cases instances, described by Riegner and Finotti, a diagnosis of the position was made from the physical signs.

C. In a third group the cyst in growing penetrates the layers of the mesocolon. Growth into the mesocolon is more likely to occur when the cyst is situated toward the tail of the gland, since this part of the organ is situated between the layers of the mesocolon; the head of the pancreas being situated in the loop formed by the duodenum, cysts from this part do not find their way into the mesocolon. Körte cites 7 instances in which the cyst situated in the mesocolon pushed forward its upper layer and reached the

anterior abdominal wall between the stomach and colon. The transverse colon may be forced downward to the pelvis, and the descending colon may overlie the tumor. In 3 instances the cyst projected upon the under surface of the membrane, so that the transverse colon was found upon its upper margin. Lazarus mentions an intermediate position in which the cyst distends the upper and lower layers of the mesocolon equally so that, as in a case of Petrykowski, the transverse colon is found to cross the summit of the tumor.

A case described by Dreyzehner, according to Lazarus,¹ does not belong to any of the groups already defined; this type may be designated prevertebral. A tumor arising from the head of the pancreas forced its way behind

FIG. 11



Cyst occupying position of mesocolon, with colon overlying. (Oser.)

the peritoneum to the right of the midline and displaced the right kidney. Payr² has described a cyst of the pancreas which was found at operation within the larger peritoneal cavity between the liver and pancreas; it had made its way through the foramen of Winslow.

Etiology and Special Pathology.—Cysts of the pancreas occur with approximately equal frequency in males and in females. Railton has described an instance of pancreatic cyst occurring in an infant six months old and Shattuck found a cyst of the gland in a boy thirteen months old.

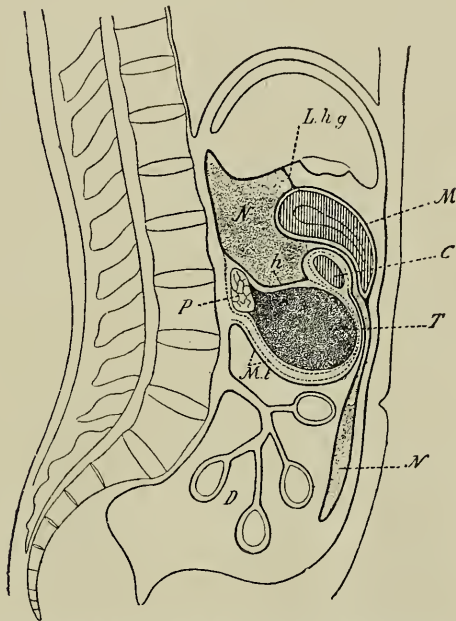
¹ *Beiträge zur Pathologie und Therapie der Pankreaserkrankungen*, Berlin, 1904, and *Zeit. f. Heilk.*, 1901, xxii, 165.

² *Wiener klin. Woch.*, 1898, xii, 629.

About one-half of the cases occur between the ages of twenty and forty years.

Retention Cysts.—Cysts formed as the result of obstruction of the larger pancreatic ducts or their branches, designated retention cysts, are often of small size and unaccompanied by clinical symptoms. Under the name of *ranula pancreatica* Virchow described dilatation of the duct of Wirsung, due in the case which he examined to occlusion caused by a villous tumor of the duodenum; the dilated duct had numerous sacculations, giving it a beaded appearance. Klebs described as *acne pancreatica* multiple small cysts containing fluid, sometimes clear and sometimes opaque yellowish and thick.

FIG. 12



Cyst projecting from lower surface of the mesocolon and presenting below the colon. (Oser.)

Ligation of the ducts of the pancreas causes only slight dilatation of the occluded channels, and is followed by chronic inflammation of the gland. Complete obstruction to the outflow of secretion in man is usually followed by similar changes. It has been thought that partial or intermittent occlusion may cause cysts. Cysts of the pancreas apparently due to retention of pancreatic secretion have been found associated with obstruction of ducts due to a variety of causes, but the conditions which favor their formation are not understood. Cysts of large size have been found in association with pancreatic calculi. Von Recklinghausen found, with a cyst almost the size of a child's head, the duct of Wirsung occluded by a calculus. Lazarus found calculi in the contents of pancreatic cysts in 2 of 14 cases, but suggests that the cyst may afford conditions which favor the formation of concretions.

Chronic pancreatitis with constriction of branches of the pancreatic duct

by newly formed strands of scar-like fibrous tissue, according to Tilger, explains the formation of many pancreatic cysts, but since chronic inflammation occurs in the wall of the distended cyst, and may be the result of compression of the larger pancreatic ducts, chronic interstitial inflammation may be the result and not the cause of cysts.

The walls of pancreatic cysts are usually several millimeters in thickness, and are composed of dense fibrous tissue, in which remains of pancreatic parenchyma may be found. The inner surface may be smooth and lined by a single layer of cylindrical cells. The presence of such cells indicates that the cyst has had its origin in a dilated duct.

Proliferation Cysts.—The occurrence of multilocular cysts of the pancreas, the presence of numerous small cysts in the wall of a large cyst, or the presence of papillary projections upon the inner surface have been regarded as evidence that cysts may be formed by proliferation of epithelial tissue of the gland. In some instances cysts of the pancreas resemble the cystadenomas of the ovary and other organs, and have been regarded as true tumors. In a case described by Riedel the entire abdomen was filled by a fluctuating tumor which during nine years had caused no symptoms. Ten liters of brown fluid were removed by puncture and the cyst was extirpated. Although the large cavity had no epithelial lining, a projection upon the inner surface contained cavities lined by cylindrical epithelium; in some of these small cysts were papillary growths resembling those found in ovarian cysts. It is not improbable that absence of epithelium in the larger cyst was the result of erosion by its contents.

Fitz¹ described a multilocular cystoma which apparently had its origin in the pancreas, and collected from the literature 9 somewhat similar cases; 8 cases occurred in women, and in most instances the growth existed for a number of years without causing severe inconvenience. In no case was there evident obstruction of ducts, but a description of some of the cysts does not exclude the possibility that they may have been due to occlusion of small ducts. Körte collected from the literature only 13 instances of proliferation cysts (cystadenomas); Munzer² (1903) cites 5 additional cases, which he places in the same group.

In two cases cysts have been associated with malignant growths. Hartmann has described as epithelioma cysticum, a tumor of the tail of the pancreas accompanied by metastases in the liver, and mentions a similar case of Hanot and Gilbert.

Traumatic Cysts.—Since many cysts of the pancreas contain blood, the relation of cyst formation to hemorrhage into the gland has been much discussed, and so-called apoplectic cysts have been described. Since, however, hemorrhage may occur into a preformed cyst, the presence of blood does not indicate that the lesion is the result of hemorrhage. Nevertheless, there is abundant evidence that injuries which may cause local hemorrhage may result in the formation of cysts. Lazarus has shown that a small hæmatoma may be formed by crushing the exposed pancreas of the dog; at the end of a week the extravasated blood may have undergone encapsulation by fibrous tissue. By crushing the pancreas of a dog he caused the formation of a hæmatoma about the size of a pigeon's egg which, after forty

¹ *American Journal of the Medical Sciences*, 1900, cxx, p. 184.

² *Cent. j. d. Grenzgeb. d. Med. u. Chir.*, 1903, vi, 490, 529, 573, 619, 664.

days was converted into a cyst with a smooth, fibrous capsule containing 100 cc. of watery fluid in which there was no blood. Lazarus thought that the hæmatoma, together with injured tissue and adjacent parenchyma, had undergone digestion by the secretion of the gland, while a reactive inflammation at the periphery of the lesion had resulted in the formation of a capsule, which in turn prevented the resorption of the fluid contents.

Among 117 cases of pancreatic cyst, Körte found 33 (28 per cent.) in which trauma had preceded the appearance of a cyst; Lazarus has collected 8 additional cases more recently published. In the greater number the epigastric region or the upper left quadrant of the abdomen has been the seat of the injury; in several cases there has been compression of the body between the buffers of two railway cars, or a wheel has passed over the body. A violent blow from the fist or the kick of a horse has preceded the appearance of a cyst. Repeated injury of less severity has been cited as the cause of the lesion; energetic massage is mentioned by Gussenbauer. In a smaller number of cases force, it has been believed, has been indirectly exerted upon the gland. Lazarus cites cases in which pancreatic cysts have followed a violent fall, as from a horse, and believes that the pancreas in such cases may be torn by the weight of the filled transverse colon dragging the movable splenic end of the pancreas away from the more firmly fixed duodenal part.

The contents of many cysts which have followed traumatism are bloody, but not infrequently such cysts have contained clear fluid free from blood. The demonstration of one or more ferments either in the contents of the cyst or in the fluid escaping from a fistula after operation has been cited as evidence of connection with the pancreas. Drainage of the cyst has been usually followed by recovery, and in only a few instances have these cysts been studied at autopsy. In the case of Richardson the pancreatic duct communicated with the cyst. Jordan Lloyd has maintained that accumulations of fluid in the lesser peritoneal cavity following contusions of the upper part of the abdomen are often mistaken for true cysts of the pancreas. In a case which he recorded fluid removed at operation had the power of rapidly converting starch into sugar, and was derived in part, he believed, from the injured pancreas.

A number of cases furnish evidence that cysts may follow acute hemorrhagic pancreatitis. The pathogenesis of the lesion doubtless resembles that of traumatic cysts. Hemorrhagic and necrotic tissue undergoes partial solution and subsequent encapsulation by fibrous tissue.

Contents.—The contents of pancreatic cysts varies greatly, being in many instances mixed with fresh or altered blood. Occasionally it is clear and watery in appearance, but more frequently the fluid is viscid, and in several instances the presence of mucus has been demonstrated. In most instances hemorrhage has occurred into the cyst, and while occasionally the contained fluid may appear to be pure blood, subsequent changes due in part to the action of digestive ferments may produce a greenish, brownish, coffee-colored, or even black fluid. Hemorrhagic cysts of the pancreas have been thought to form a separate group, and their bloody contents have been regarded as indication of traumatic origin. On the one hand, cysts following traumatism have contained clear, watery, or yellowish fluid, since partial digestion and absorption of blood may remove all evidence of its former presence. On the other hand, retention cysts and other cysts with

which there is no history of injury to the gland may contain bloody fluid. The numerous and often congested vessels in the walls of many cysts are the origin of hemorrhage, and, it is believed, are not infrequently eroded by the contents of the cavity.

Microscopic examination of the cystic contents gives little evidence of the character of the lesion, and may show the presence of epithelial cells, leukocytes, often containing fat, globules of fat, crystalline fatty acids, altered red blood corpuscles, and necrotic tissue.

Much significance was formerly attached to the presence of ferments resembling those of the pancreatic secretion, but the absence of one or more of these ferments in the contents of pancreatic cysts and the presence of similar ferments in cysts of other organs has greatly diminished their diagnostic value. The presence of tryptic, diastatic, and emulsifying ferments may suggest the character of fluid removed by operation, but rarely is it possible to demonstrate all three ferments, while most frequently the diastatic ferment alone can be found. The antitryptic action of the blood serum may explain occasionally the absence of trypsin in cysts with hemorrhagic contents. Only when the cyst is in communication with the secreting parenchyma can the ferments of the gland find their way into its contents, while observation on animals (Heidenhain) shows that the secretion of the chronically inflamed pancreas may contain little, if any, ferment. The secretion of a fistula formed as the result of a pancreatic cyst contains ferments more frequently than the contents of cysts.

Fat-splitting, diastatic, or proteolytic ferments, moreover, have been demonstrated in a variety of fluids which do not have their origin in the pancreas. Since Körte has found a weak diastatic ferment in the contents of a mesenteric cyst, he thinks that fluid from a cyst must exhibit active diastatic action in order to suggest a probable pancreatic origin. Boas thinks that tryptic ferment is characteristic of these cysts, but Lazarus has found a fibrin-digesting ferment in the contents of an ovarian cyst, and it is probable that any fluid containing leukocytes in considerable number will exert proteolytic action in an alkaline medium.

Symptoms.—A rounded *tumor* fluctuating on palpation and situated in the epigastric region behind the stomach and a greater part to the left of the median line suggests the presence of a pancreatic cyst, but the variation to which these characters are subject often makes recognition of the tumor difficult. Small cysts which have caused no palpable tumor and no recognizable symptoms are occasionally found at autopsy, while, on the other hand, a cyst of great size, completely filling the abdominal cavity, has been thought to arise from one of the pelvic organs. In the majority of cases the tumor is situated between the ensiform cartilage and the umbilicus, and may produce a rounded protrusion of the abdominal wall. In 48 cases collected by Körte the tumor occupied the median line; in 40 instances the greater part of the mass was to the left of the median line, extending to the left costal margin, and in some instances into the lumbar region. In only 10 cases was the larger part of the cyst to the right of the median line. Very large tumors extend into the lower part of the abdominal cavity, and in 16 cases the tumor extended below the level of the umbilicus, sometimes as far as the symphysis pubis.

The size of the cyst is subject to equal variation; in many instances it is described as the size of a man's head, but may distend the abdominal cavity,

extending from the ensiform process to the symphysis pubis. In shape the cyst is usually spherical and the surface is smooth. Fluctuation can usually be felt, but rarely the fluid contents have distended the sac so tensely that the tumor has appeared to be solid. When the cyst is grasped between the hands, one placed upon its abdominal surface, the other upon the lumbar region, pressure of one hand is transmitted to the other. Pancreatic cysts, contrary to the statements of some writers, are not infrequently movable, and, particularly when in contact with the diaphragm, move with respiration. Cysts arising from the tail of the gland are particularly movable, and unless fixed by adhesions can be pushed both from above down and laterally. In a case described by Lazarus a fluctuating tumor the size of an infant's head was situated in the left hypochondrium, but could be pushed almost to the right mammillary line. Cysts which lie in front of the aorta may, when sufficiently tense, transmit its pulsation, but cease to pulsate when the patient is in the knee-chest position.

Of much importance is *the relation of the cyst to adjacent organs*, and especially to the stomach and colon, between which in most instances it reaches the abdominal wall. Small cysts may be wholly covered by the stomach, but with increase of their size the organ is pushed upward, so that when artificially distended its tympany separates the dulness produced by the cyst from that of the liver. The colon, best mapped out after inflation with air, is found along the lower border of the cyst, being in some instances pushed downward to the symphysis pubis. When the cyst occupies the mesocolon, the colon may be below the tumor, may cross its summit, or lie along its upper border in accordance with the previously described relation which the cyst bears to the upper or lower surface of the membrane. When the cyst projects upon the lower surface of the mesocolon, the tumor may occupy the lower part of the abdominal cavity, with its margin in contact with the symphysis pubis. Cysts which force their way between the liver and the stomach produce dulness in contact with that of the liver while their lower part is covered by the stomach, which, when distended, almost completely covers the tumor.

Sudden *disappearance of the tumor* caused by a pancreatic cyst may be the result of rupture into the peritoneal cavity. In some instances rupture has followed puncture undertaken for the purpose of diagnosis, or, as in the cases of Halsted and of Schwartz, has followed injury the result of a fall. Of greater interest is the spontaneous disappearance which has been accompanied by temporary diarrhoea of a peculiar character, suggesting that the contents of the cyst have been discharged into the duodenum. Disappearance of the tumor in the cases of Bull and of Karewski was accompanied by the appearance of dark-colored masses in the stools. In the case described by Parson the sudden disappearance of a tumor the size of an orange situated in the epigastrium was followed by profuse whitish, diarrhoeal discharge; a second tumor appeared in the lower part of the abdomen at the end of several weeks, disappeared with profuse diarrhoea, and again returned. A large cyst in the epigastrium described by Payr disappeared with occurrence of diarrhoeal discharges containing whitish particles three times during two months. A communication between the cyst and the ducts of the gland may explain the disappearance of such tumors, but the conditions which cause the periodic discharge of the cystic contents have not been explained.

Pain is one of the most constant symptoms, and is not infrequently present before a tumor is demonstrable. In a few instances the presence of the tumor has been unaccompanied by pain, or has merely caused a feeling of distention. Pain is usually deeply seated in the epigastric region, or may be referred to the small of the back. In some instances it has been referred to the right or left hypochondrium in accordance with the localization of the cyst in the head or tail of the gland, but little reliance can be placed upon these subjective symptoms. The intensity of the pain varies greatly, it may occur in paroxysms which in some instances have the character of gastric crises, and may be accompanied by vomiting and collapse.

A variety of symptoms have been produced by the *pressure of the growing cyst upon adjacent organs*. Most constant are those referable to the stomach, which in some instances is flattened by the tumor. Discomfort after eating, loss of appetite, vomiting, and other symptoms of disturbed digestion are present, and may have long preceded other evidence of disease. Compression of the intestine may be accompanied by colicky pain and constipation. In a case of Lazarus the transverse colon situated along the lower margin of the tumor was frequently the seat of colic, and in four cases collected from the literature by this author complete intestinal obstruction had occurred.

Cysts situated in the head of the pancreas may cause *jaundice*. The relative infrequency of this symptom is explained by the fact that cysts, unlike carcinoma, occur more frequently in other parts of the gland. Retention cysts caused by tumors or by biliary calculi compressing the pancreatic duct are accompanied by jaundice due to the same cause.

Compression of the portal vein may be followed by *ascites* and by dilatation of its branches or of the collateral circulation. Dilatation of the mesenteric veins and vessels upon the surface of the cyst adds to the danger of puncture. Compression of the vena cava has been accompanied by œdema of the lower extremities. In two instances (Dreyzehner, Reeve) one ureter has been obstructed.

Functional disturbance of the pancreas has been noted with only a small proportion of the cysts which have been described, but careful chemical observation of the feces and tests for alimentary glycosuria would probably disclose their presence in a larger proportion of cases. Cysts situated in the body or tail of the gland do not so readily cause such disturbances as the rarer cysts which arise from the head of the gland, since they do not occlude the large duct, and therefore do not cause chronic inflammation of the entire gland. Among 29 instances of *steatorrhœa* accompanying well-authenticated disease of the pancreas collected by Fitz the symptom was referable to cysts in only 2 cases (Bull, Goodman), in both of which diabetes was present as well. The same author found 2 cases in which, with pancreatic cysts, disturbance of proteid digestion was indicated by the presence of undigested muscle fibers in the feces, and additional cases have been described by Kuster and Lazarus.

Diabetes mellitus results from the presence of cysts only when the gland is the seat of advanced chronic interstitial inflammation. Diabetes is, however, rarely present, and occurred only in 9 of 134 cases collected by Oser. Alimentary glycosuria in association with pancreatic cyst has been observed in a case described by Lazarus.

Loss of weight and weakness commonly accompany pancreatic cysts, and may be referred in part to the digestive disturbances and vomiting

caused by the pressure of the tumor. Lazarus thinks that the cause of cachexia is more obscure, and in several instances has seen loss of weight, although there was no disturbance of digestion, no diabetes, and no jaundice. One of his patients, a healthy man, who developed a cyst in consequence of traumatism, became much emaciated, but after operation recovered so quickly that within six weeks he had regained fifteen pounds.

Diagnosis.—Recognition of pancreatic cysts depends almost wholly upon physical signs, although in a comparatively small number of cases diabetes or steatorrhœa confirms the diagnosis. The presence of fluctuation may serve to distinguish cysts from solid tumors of the pancreas or of retroperitoneal structures, but with cysts so tensely distended that this sign is absent some uncertainty may exist, although the spherical shape and smooth surface may indicate the character of the lesion. In determining the origin of such a tumor, of greatest significance is its relation to the stomach and colon, behind which it is situated. This is done best by percussion after inflation. In some instances information is obtained by noting the change in position of the stomach during the progress of inflation; as the organ enlarges, it gradually overlaps an increasing area at the upper or lower margin of the tumor, in some instances completely covering it. The difficulty of diagnosis is greatest with those unusual cysts which present above the stomach or along the lower margin of the colon, the typical position being between stomach and colon and behind both.

Possible etiological factors may have an important bearing upon diagnosis. A history of injury in the epigastric region may suggest the presence of pancreatic cyst of traumatic origin. Symptoms of hemorrhagic pancreatitis may precede the appearance of a tumor mass in this region.

Since cysts of the pancreas may occupy almost any part of the abdominal cavity, their differential diagnosis presents many difficulties. Cysts may occur in the liver, spleen, mesentery, omentum, ovaries, retroperitoneal tissue, and adrenals, while a distended gall-bladder or hydronephrosis may resemble a cyst of the pancreas.

Echinococcus cysts of the left lobe of the liver may form large, fluctuating tumors in the epigastric region. The dulness produced by pancreatic cysts occupying their usual position between the stomach and colon is separated by the tympany of the former organ from the area of hepatic dulness, but should the cyst present between liver and stomach, diagnosis is more difficult. The stomach, particularly when distended, is found in front of the pancreatic tumor, which is pushed backward and becomes less palpable, but the organ cannot bear the same relation to a hepatic cyst which lies in contact with the abdominal wall. Should the cyst be punctured, the well-known hooklets, or perhaps particles of the chitinous layer of the cyst wall, may be found in an echinococcus cyst. The *distended gall-bladder*, situated in the usual position of this organ, has the respiratory movement of the liver, and, lying in contact with the abdominal wall, is rarely covered by the stomach or intestine. Cysts of the head of the pancreas, suggesting a distended gall-bladder, may be accompanied by jaundice.

Cysts of the mesentery have usually been found in the neighborhood of the umbilicus, and, unless fixed by adhesions, are movable in both vertical and horizontal directions. Cysts of the mesocolon are indistinguishable from similarly situated cysts of the pancreas.

The diagnosis between *ovarian cysts* and pancreatic cysts is in most

instances made with little difficulty. Pancreatic cysts which arise from the tail of the gland and are situated in the mesocolon lie in contact with the pelvis and may closely simulate ovarian cysts, while cysts of great size distending the abdominal cavity may offer the same difficulty. In some instances the tumor has been first observed in the epigastric region, from which it has subsequently extended downward. Bimanual examination by vagina and rectum of an ovarian cyst may demonstrate a pedicle connecting it with the uterus and perhaps dragging the latter upward. With pancreatic cysts the uterus occupies its normal position, and the ovaries are often palpable. Pancreatic cysts of great size are situated behind the stomach and colon, while ovarian cysts push these organs upward.

Pancreatic cysts may closely simulate *hydronephrosis* affecting the left kidney; the retroperitoneal cyst described by Dreyzehner arose from the head of the pancreas, and, displacing the right kidney, resembled hydronephrosis on this side. The history indicating that the tumor has had its origin in the epigastrium may suggest pancreatic cyst. The tumor of hydronephrosis fills the lumbar region and can be forced even farther backward by distending the colon; pancreatic cysts, as a rule, do not extend beyond the axillary line behind which there is tympany. Periodic disappearance of the tumor may occur both with hydronephrosis and with pancreatic cyst, but abundant flow of urine or simultaneous onset of diarrhoea may suggest one or other condition.

Since pancreatic cysts when tensely distended have been known to transmit pulsations from the aorta, *aortic aneurism* may be suspected. The tumor rises and falls, but has no expansile pulsation; in the knee-chest position pulsation disappears.

Treatment.—Cysts of the pancreas have been treated by puncture, by extirpation, and by incision and drainage. Puncture followed by aspiration has proved an ineffectual and dangerous procedure. Kuster and Lazarus have mentioned the occurrence of hemorrhage into a cyst due to puncture of enlarged vessels in the wall. When the cyst is infected, purulent, general peritonitis may follow puncture. Removal of cystic contents by aspiration is usually followed by re-accumulation of fluid, even when the cyst is of traumatic origin and has walls which are not lined by epithelium. In a case described by Steele puncture was followed five times by reappearance of the cyst. In a case of Israel¹ laparotomy disclosed a cyst completely covered by the stomach; twelve days later the cyst was punctured directly through the stomach, more than a liter of brown fluid was removed and the cyst did not reappear. Such a favorable result occurs very rarely.

Complete extirpation of a pancreatic cyst was successfully performed in 1882 by Bozemann. The operation is difficult and dangerous, on account of the close proximity of important bloodvessels, and may be followed by diabetes mellitus when a considerable part of the gland is removed. The operation is rarely practicable, unless the cyst has a well-defined wall, and is therefore not applicable to traumatic cysts and other so-called pseudocysts. In several instances complete extirpation has been performed after the multilocular character of the cyst or the presence of papillary projections upon its inner surface have suggested the existence of an adenomatous growth. Complete removal of these proliferation cysts has been

¹ *Deutsch. klin. Woch.*, 1900, xxvi, 352.

believed to offer the best chance of avoiding recurrence, although in several instances incision and drainage of cysts believed to belong to this type have been followed by recovery. A fatal result is due to gangrene of the intestine, hemorrhage, or peritonitis; when dense adhesions, the proximity of blood-vessels, or the deep situation of the tumor has prevented the completion of attempted extirpation, the mortality has been greater. When a cyst occupies the usual position between stomach and transverse colon, it is in most instances bound by adhesions to adjacent structures, and removal is difficult; when, however, the cyst lies within the mesocolon, adhesions are usually less abundant, and a considerable proportion of cysts which have been extirpated have had this situation. The operation is least difficult when the cyst occupies the tail of the gland, which serves as a pedicle and allows the tumor to be drawn into the wound; it is more difficult the nearer the cyst approaches the duodenum.

When the cyst has been partially removed the wound has been drained and healing has occurred by granulation. In most instances of complete extirpation, pancreatic fistula has been avoided, but in two cases cited by Körte pancreatic secretion discharged through the wound. Zweifel removed the entire pancreas save a part of the head 3 cm. in length; glycosuria was present ten days later, and persisted about twelve days, but at the end of two months had disappeared.

The disadvantages of incision and drainage are the occurrence of pancreatic fistula persisting for a considerable period, the possibility of hernia at the site of operation, and the occasional recurrence of the tumor. The resulting wound often heals slowly, and is complicated by the formation of a pancreatic fistula, from which there may be copious secretion; in the case of Cushing from 500 to 600 cc. of fluid were discharged during twenty-four hours. Pancreatic ferments may be demonstrable in the secretion from the wound immediately after operation, or may make their appearance several weeks later; unless care is exercised, there may be erosion of the skin about the wound. The cyst walls collapse, and by formation of granulations from the surrounding connective tissue the cavity is finally obliterated. This process cannot be completed when the cyst has an epithelial lining.

A fistula may persist a considerable time after operation; in a case described by Murray fistula was present during three years. In a case of Körte a fistula persisted two and a half years; various substances such as tincture of iodine, silver nitrate, and chloride of zinc, were injected into it, and the wall was scraped and cauterized in the hope that perhaps an epithelial covering which prevented healing might be destroyed. The danger of injecting irritant substances into a fistulous tract is illustrated by a case described by Lazarus; death with collapse, due perhaps to acute hemorrhagic pancreatitis, followed the injection of silver nitrate. Infection of a fistulous tract may occur. In some instances there has been hemorrhage from a fistula; curetting of the tract is attended with danger, since dilated bloodvessels may be present in the wall. Closure of a pancreatic fistula has been hastened by making a second posterior opening, thus affording better drainage, or rarely by extirpation of the entire tract.

Healing of a pancreatic cyst is associated with the formation of scar tissue in the pancreas, and chronic inflammation resulting from the presence of the cyst may persist. In four cases in which recovery has followed operation diabetes mellitus has resulted.

CARCINOMA OF THE PANCREAS.

Recent study of pancreatic disease does not confirm the statement of Friedreich that cancer is the most frequent form of primary disease of the gland. Carcinoma is more frequent than other forms of neoplasm.

Incidence.—In the statistics of Bashford malignant growth was primary in the pancreas in males in 526 among 33,788 instances of cancer, and in females in 474 among 50,660 cases. In the statistics of Segré, among 11,472 autopsies, there was carcinoma of the pancreas in 127 and sarcoma in 2 instances. In the older literature of the subject chronic interstitial inflammation with induration of the gland has been not infrequently designated scirrhus cancer. The disease affects men more frequently than women. Among 106 reported cases of primary cancer collected by Mirallié¹ in 1893, 69 were in men and 37 in women, the disease being almost twice as frequent in men as in women. Cancer of the pancreas like that of other organs usually occurs during middle life, although there have been instances of its appearance at a very early age. Kuhn has described carcinoma of the pancreas in a child two years old, and Simon found the same disease in a boy thirteen years of age.

Secondary Carcinoma.—Since the pancreas is surrounded by organs which may be the seat of primary new-growth, it is often difficult, when the tumor has attained considerable size, to determine whether it is primary in the pancreas or has invaded it from without. Metastatic tumors occasionally occur in the pancreas, but the organ is more frequently the seat of secondary invasion by cancer of the stomach, duodenum, biliary passages, or other organs. Since the pyloric region and the posterior wall of the stomach are the most frequent situations of gastric cancer, secondary involvement of the head of the pancreas readily occurs, and is followed by chronic interstitial inflammation of the body and tail as the result of compression of the duct of Wirsung. Fährdrich maintains that carcinoma supposed to be primary in the pancreas has its origin in a considerable number of instances in the mucosa of the stomach, the two organs being affected almost simultaneously. Olivier has described adenocarcinoma occupying the head of the pancreas, apparently originating from Brunner's glands of the duodenum. He believes that careful microscopic examination is necessary to determine the origin of many tumors which are apparently primary in the pancreas.

Special Pathology.—Carcinoma of the pancreas is much more common in the head than in other parts of the gland. Among 57 cases collected by Segré, the tumor in 19 instances occupied the whole gland, in 35 the head, in 2 the body, and in 1 the tail. In the cases of primary cancer collected by Marallié the head of the pancreas was affected in 82 of 113 cases. Hanse-mann has described a type of pancreatic cancer which affects diffusely the whole gland. In most cases alveoli of cancer cells are embedded in abundant, dense, fibrous stroma; the tumor is hard and of the scirrhus type. Less frequently the neoplasm is cellular and soft or encephaloid; several cases of colloid carcinoma have been described. A cystic epithelioma is described by Roux.

¹ *Gaz. des Hôpitaux*, 1893, lxvi, 889

Since the pancreas contains several distinct structures, namely glandular alveoli, ducts, and islands of Langerhans, the attempt has been made to determine the histogenesis of different types of new-growth. In most instances epithelial neoplasms of the pancreas are adenocarcinomas, being composed of cylindrical cells arranged in more or less regularly formed tubular alveoli. These tumors, according to Hulst, are probably derived from the ducts of the gland. Where the glandular type is absent and alveoli of the tumor are formed by irregular polygonal cells closely packed together, Hulst¹ thinks it more probable that the tumor has its origin in the secreting alveoli. Nevertheless, in a case carefully studied by Olivier a tumor of the tail of the gland which in part exhibited the last named type could be traced to the ducts, with which alveoli of cancer cells were in places continuous. Fabozzi, from a study of 5 cases of this kind, designated by him *carcinoma solidum*, reaches the conclusion that such tumors are derived from the islands of Langerhans, but Hulst, on the contrary, found no relation between the islands of Langerhans and the alveoli of tumor cells. Ssbolew has suggested the possibility that a cancer derived from the islands of Langerhans might exert upon carbohydrate metabolism that influence which is attributable to the normal gland, and might prevent the occurrence of diabetes even though the entire gland were destroyed.

Adenoma.—Adenoma, unassociated with cyst formation, occurs rarely, and in most instances found at autopsy has given no discomfort during life. An exception is the encapsulated tumor of the head of the pancreas described as fibro-adenoma by Biondi; it was palpable and caused slight jaundice. Nicholls described a tawny yellow tumor which was the size of a large pea and had the structure of the island of Langerhans; a similar tumor is described by Helmholtz.

Sarcoma.—Sarcoma of the pancreas occurs with far less frequency than carcinoma. Kakels,² in 1902, collected 21 instances, among which only 10 were certainly primary in the gland; in 3 of the latter the tumor occupied the tail of the pancreas. Lymphosarcoma, angiosarcoma, fibrosarcoma, and medullary sarcoma have been described. The age of those affected has been of little value for diagnosis, since it has varied from four to seventy-four years. In a case described by Malcolm, not included among those just mentioned, a tumor five inches in diameter was removed from the tail of the pancreas in a child four years of age; at autopsy the growth was found to have invaded the portal vein.

Pathology of Complications.—Since *diabetes mellitus* not infrequently occurs in association with carcinoma, and is due to complete or partial destruction of the organ, the changes in the gland caused by the presence of the new-growth are of considerable importance. Compression of the duct of Wirsung caused by the tumor mass, which is usually situated in the head of the gland, is followed by chronic interlobular inflammation of that part of the gland from which the outflow of secretion is hindered. The secreting parenchyma is destroyed, while the islands of Langerhans tend to persist in the newly formed fibrous tissue. Pearce³ has studied the changes caused by the presence of carcinoma. At the edge of the advancing cancer the secreting parenchyma is invaded and replaced by new-growth and newly formed

¹ *Virchow's Archiv*, 1905, clxxx, 228.

² *American Journal of the Medical Sciences*, 1902, cxxiii, 471.

³ *Ibid.*, 1904, cxxviii, 478.

connective tissue, but the islands of Langerhans persist in the stroma, and occasionally are uninvaded, even though enclosed by a mass of tumor cells; much hypertrophied islands are found. Carcinoma with permanent glycosuria was present in only one case studied by Pearce, and was associated with advanced chronic inflammation, implicating the islands of Langerhans. In a second case with intermittent glycosuria the head of the gland alone was the seat of new-growth.

As the tumor increases in size, adhesions may bind it to the stomach, duodenum, and adjacent organs, and symptoms are frequently caused by *pressure of the enlarging mass*. The following possibilities are named by Friedreich, who cites cases in illustration of each. There may be closure of the duodenum or of the pylorus, with consequent dilatation of the stomach; occasionally the cardiac end of the stomach is compressed. In one case the stomach was compressed against the anterior abdominal wall. Compression of the transverse colon may cause symptoms of intestinal obstruction. Carcinoma affecting the tail of the pancreas has compressed the left ureter, causing hydronephrosis. Compression of the common bile duct causes jaundice. The growth may compress the superior mesenteric artery and vein, or the splenic artery and vein; pressure upon the portal vein causes ascites, while pressure upon the inferior vena cava causes oedema of the lower extremities. Pulsation of the aorta may be transmitted to the abdominal wall. Necrosis and ulceration may occur in tumor tissue which has invaded adjacent organs, and ulceration of the stomach or of the duodenum may cause the symptoms which are usually associated with primary cancer of these organs.

Symptoms.—The symptomatology of primary carcinoma was first carefully investigated by Bard and Pic, who described a combination of symptoms believed by them to be characteristic. Their description was based upon seven cases, in all of which the tumor occupied the head of the gland, and in consequence the symptoms, which they regarded as characteristic, were the result of compression of the bile duct. In such cases jaundice is present, and, being due to progressive compression of the duct, never recedes, but gradually increases in intensity until finally it merits the designation "black jaundice"; the gall-bladder is distended and is palpable in the usual situation. Hepatic metastases secondary to pancreatic cancer, according to Bard and Pic, are of small size, and do not cause that enlargement of the liver which is associated with primary hepatic carcinoma or with carcinoma having its origin in the gastro-intestinal tract. The temperature is normal or subnormal; the disease pursues a rapidly fatal course, and is accompanied by emaciation and cachexia.

It is now well established by the statistical studies of Mirallié and others that primary cancer of the pancreas does not always present the characteristic symptomatology described by Bard and Pic. Cancer of the body or tail of the gland may be unaccompanied by jaundice and other symptoms referable to compression of the biliary passages. Moreover, the liver may be enlarged so that in the presence of jaundice the size of the liver will not serve to exclude cancer having its primary origin in organs other than the pancreas. Contrary to the opinion of Bard and Pic, cancer of the pancreas, like cancer of the other organs, is subject to great variation in the rapidity of its growth, death occurring within from a few months to two or very rarely even four years after the onset of symptoms.

Pain is one of the earliest and most constant symptoms, and is usually seated in the epigastric region radiating toward the back, shoulders, or sternum; the right hypochondrium may be the seat of greatest pain when the tumor is situated in the head of the gland. The character and severity of the pain varies considerably; it may continuously increase in intensity until death, and may be of the intense severity which has been referred by many writers to pressure upon the celiac ganglion and its branches. Intermittent colicky pain has been regarded as true pancreatic colic due to obstruction to the outflow of the pancreatic secretion caused by compression of the pancreatic duct. Similar pain may be the result of compression of the bile passages.

A *tumor mass* has been palpable in a relatively small proportion of the reported cases. Mirallié found that the tumor was palpable in from one-fourth to one-fifth of 113 cases which he collected. In the statistics of Ancelet, from which secondary tumors of the pancreas were not excluded, a tumor was present in 36 in the epigastrium, in 4 in the right, and in 2 in the left hypogastrium, while in 4 its situation was not accurately designated. The tumor must be of considerable size to be felt, especially when the abdominal wall contains abundant fat or is muscular; extreme tenderness may interfere with palpation. Although the tumor is usually immovable, it may descend with inspiration, and occasionally transmits pulsation from the aorta. In some cases the distended gall-bladder has been mistaken for a tumor mass, while at times both tumor and enlarged gall-bladder have been distinguishable.

Symptoms referable to the *gastro-intestinal tract* not infrequently precede more definite evidence of pancreatic carcinoma. There may be lack of appetite or actual distaste for food, especially, it has been thought, for meat. There may be discomfort after eating, eructation, nausea, or vomiting. Compression of the pylorus or of the duodenum may cause dilatation of the stomach, and in some instances symptoms referable to this condition have been the only evidence of the disease (Pilliet). The vomitus in such cases may contain blood. Constipation has been found much more often than diarrhoea, while constipation and diarrhoea may alternate; the stools may be normal.

Disturbance of digestion referable to lack of pancreatic juice in the intestine has been noted more frequently with carcinoma than with other diseases of the gland; nevertheless, Mirallié found only 9 cases with *fatty stools* among 113 instances of pancreatic carcinoma. Oser has collected 8 and Fitz 3 additional cases from the literature of the subject. Fitz found only 2 cases in which cancer of the pancreas was accompanied by disturbed digestion of proteids manifested by the presence of numerous undigested muscle fibers in the fæces. Bulky stools are believed by Oser to be a symptom of pancreatic disease, and occur as the result of imperfect digestion of fats, proteids, and carbohydrates.

Since carcinoma of the pancreas in a large proportion of the cases—at least 61 of 78 cases, according to Mirallié—invades the head of the gland, *jaundice* due to compression of the common bile duct is a frequent symptom, occurring in three-fourths of the cases, and since the lesion in a large proportion of the cases has its origin in this part of the gland, being limited to the head in 39 of the 78 cases just cited, frequently occurs as one of the earliest symptoms. Moreover, carcinoma of the pancreas, save for chole-

lithiasis, is the most frequent cause of occlusion of the common bile duct, and has been the cause of obstruction in 54 of 139 cases of non-calculous occlusion cited by Ecklin.¹ Jaundice due to compression of the duct may appear suddenly, and, once established, exhibits no remission, but steadily increases in intensity. When its sudden appearance is accompanied by colicky pain, cholelithiasis is closely simulated.

The condition of the *gall-bladder* and of the liver with obstruction of the common bile duct by cancer of the pancreas has been much discussed. Battersby and Bard and Pic pointed out that the gall-bladder is dilated with cancer of the pancreas. From a study of a considerable number of cases of obstruction of the common bile duct due to a variety of causes, Courvoisier found that with obstruction due to biliary calculi the gall-bladder is usually contracted, while with occlusion due to other causes the gall-bladder is usually dilated. The statistics collected by Ecklin confirm this view. Among 172 instances of obstruction of the common bile duct due to gallstones, the gall-bladder was contracted in 110, normal in 34, and dilated in 28 cases. On the other hand, among 139 instances in which the common bile duct was obstructed by other causes, the gall-bladder was contracted in only 9, normal in 9, and dilated in 121 cases; among 62 of these cases constriction of the common bile duct was due to carcinoma of the pancreas, and the gall-bladder was dilated in 58 instances. Contraction of the gall-bladder with biliary calculi is the result of inflammatory changes, whereas, with obstruction due to carcinoma of the pancreas or other cause, inflammation is usually absent and the gall-bladder is dilated.

Bard and Pic attached much importance to absence of enlargement of the *liver* with carcinoma of the pancreas. Mirallié, nevertheless, found the liver enlarged in 17 cases, and a case in which enlargement of the organ disappeared under observation led him to believe that the liver is at first enlarged and later contracted, its size depending upon the duration of the disease. Oser cites the cases of Friedreich and of Kellermann, in which, contrary to the statement of Bard and Pic, the liver contained, during the terminal stage of the disease, secondary nodules as large as an apple, and maintains that the liver with cancer of the pancreas has no constant character.

Compression of the portal vein may cause *ascites* and other evidences of portal obstruction, such as swelling of the spleen and hemorrhoids. In 13 of 113 cases of primary carcinoma collected by Mirallié, ascites was present. Cases with chylous ascites presumably due to rupture of the thoracic duct have been described.

Glycosuria and other symptoms of *diabetes mellitus* not infrequently accompany cancer of the pancreas; among 50 cases Mirallié has found glycosuria in 13, and Oser has subsequently been able to collect 8 additional instances of persistent glycosuria and 2 of alimentary glycosuria. Diabetes was present in 2 cases described by Pearce, and alimentary glycosuria in 1. Cancer of the pancreas is an infrequent cause of diabetes. Islands of Langerhans persist within the stroma of the new-growth and prevent the onset of glycosuria until destruction of the pancreas is far advanced.

The glycosuria of pancreatic cancer, like that of other forms of diabetes, may, as Mirallié has pointed out, disappear before death. Courmont and Bret have described such a case in which ten weeks after the onset of symp-

¹ *Inaug. Diss.*, Basel, 1896.

toms 2.6 per cent. of sugar was found in the urine, but with increasing jaundice and cachexia glycosuria disappeared.

The *cachexia* usually associated with malignant growth is said to proceed with especial rapidity when the pancreas is the seat of the disease. Partial exclusion of pancreatic juice from the intestine and obstruction to the outflow of bile doubtless contribute to cause weakness and emaciation. Kellermann, who has described a case of cancer of the pancreas unaccompanied by cachexia, has been able to find only three similar cases in the literature of the subject.

The *temperature* with carcinoma of the pancreas is normal or subnormal unless there are complications accompanied by infection.

Diagnosis.—For this the features of greatest importance are jaundice increasing gradually, attaining great intensity, and associated with dilatation of the gall-bladder, the presence of a tumor in the epigastric region, rapid emaciation, and advanced age. In the absence of both tumor and jaundice diagnosis is hardly possible. Symptoms indicative of impaired pancreatic function are present in a relatively small proportion of the cases, but when present give definite evidence that the disease is located in the gland.

When jaundice is present, differential diagnosis between *cholelithiasis* and carcinoma is important. In certain cases of pancreatic carcinoma the biliary symptoms described by Bard and Pic serve to distinguish the two lesions. Jaundice usually appears gradually with carcinoma and increases without remissions, while with biliary colic the onset is more sudden and the progress more interrupted. Impacted calculi may, however, cause progressively increasing jaundice of great intensity; the gall-bladder is dilated in only a sixth of the cases, but with carcinoma of the pancreas, on the contrary, the gall-bladder is with few exceptions dilated. A palpably enlarged gall-bladder, therefore, gives evidence that cholelithiasis is not present, but the absence of this symptom is not equally significant, since the dilated organ is not always palpable, particularly when the abdominal wall contains abundant fat. Rapid emaciation and intense epigastric pain point to cancer of the pancreas. With carcinoma the temperature is usually normal or subnormal, but with cholelithiasis fever is not infrequently present.

Jaundice caused by *carcinoma of the liver, bile passages, duodenum, or stomach, compressing the common bile duct*, has the gradual onset and progressive character already mentioned, but emaciation and weakness are said to occur less rapidly than with carcinoma of the pancreas. Metastatic nodules secondary to cancer of the pancreas, according to Bard and Pic, do not cause the enlargement of the liver which accompanies other forms of hepatic cancer, but exceptions are not infrequent. Tumor has been recognized in a relatively small proportion of cases of cancer of the pancreas, but doubtless this proportion would be much increased by examination under anæsthesia with the stomach and colon empty. A tumor mass having its origin in the head of the pancreas is deeply situated in the epigastric or umbilical region, and is usually defined with difficulty. The tumor is usually less movable than tumors of the pylorus or of the colon, and its position is unaffected by distention of these organs. Pulsation transmitted from the aorta is not expansile. It has been mentioned that both dilatation of the stomach and intestinal obstruction may be caused by carcinoma of the pancreas. The differential diagnosis between primary carcinoma of the

pancreas and malignant growth arising from the common bile duct or duodenum is scarcely possible, and may be doubtful even at autopsy.

Tumor mass due to *carcinoma of the tail of the pancreas* may be mistaken for cancer of the cardia or of the colon. Takayasu describes a case in which an apparently immovable tumor, the size of a hen's egg, was felt behind the left rectus muscle. With inflation of the stomach, which lay above it, the tumor partially disappeared, and with inflation of the colon, which was at its lower margin, disappeared completely. Operation confirmed the diagnosis of carcinoma of the pancreas.

Treatment.—Medical treatment is directed to alleviation of symptoms, such as pain, jaundice, digestive disturbances, and diabetes. The administration of an emulsion prepared from the fresh pancreas of the pig has been found to aid digestion of fats and of proteid when obstruction of the pancreatic ducts prevents entrance of pancreatic juice into the intestine (Fles); for the same purpose commercial pancreatin which contains both trypsin and steapsin has been recommended.

In a small number of cases tumors of the pancreas have been removed by *operation*; in a considerable proportion of the cases the patient has survived the immediate effects of the operation. Of 13 cases collected by Körte and by Mayo Robson, 5 died as the immediate result of the operation, whereas 5 are known to have died within from a few weeks to five months after the operation. In one instance (Biondi) a tumor, palpable on the left side of the abdomen, was removed from the head and body of the pancreas, and was found to be a fibro-adenoma; one and a half years later the patient was well. It is noteworthy that among the tumors which have been removed those types which are found least frequently have been relatively common; sarcoma has occurred three times, and in six cases the growth arose from the tail of the gland, causing a palpable tumor on the left side of the abdomen. Franke¹ believed that he had removed the entire pancreas, but its complete absence was not confirmed by autopsy; sugar was found in the urine from the fifth to the nineteenth day after operation, and death occurred at the end of five months.

For the *relief of complications* a variety of palliative operations have been performed, but it is doubtful that they have materially prolonged life or added to the comfort of the patient. When jaundice due to compression of the common bile duct has caused much discomfort, cholecystenterostomy has been performed with the purpose of allowing the bile to escape and at the same time permitting its return to the intestine. In several instances the patient has lived from six to eighteen months, and the condition apparently has been improved. Weakness and intense jaundice increase the dangers of shock and hemorrhage. Little improvement has followed the formation of an external biliary fistula, and death has usually occurred a short time after operation (Nimier). When persistent vomiting and dilatation of the stomach indicate compression of the pylorus or of the duodenum, gastroenterostomy may give relief, but since the biliary and pancreatic ducts are usually compressed, the operation has not the advantages which it offers for the relief of the symptoms of pyloric carcinoma.

¹ *Verhandl. d. Deutschen. Gesell. f. Chir.*, xxx, Congress, 1901, 265.

CHAPTER X.

DISEASES OF THE LIVER, GALL-BLADDER, AND BILIARY DUCTS.

By A. O. J. KELLY, M.D.

THE LIVER.¹

DISORDERS OF FUNCTION.

THE liver, the largest glandular organ in the body, is of complex structure and is endowed with a multiplicity of functions. Several of the structural peculiarities are mentioned in connection with the discussion of the histology of cirrhosis, and the structural changes that occur in different diseases are discussed under the respective headings. The disorders of the functions of the liver are less well understood.

Inasmuch as health and disease each consists of a sum of variables that have no well-defined limits, overlap materially, and pass gradually the one into the other, it is not surprising that the ultimate beginnings of disease often escape our observation. As regards the liver specifically, if, as for instance in certain infections, the time of the onset of the infection is apparently quite obvious, in many other diseases, especially those of a more

¹ The older views in regard to diseases of the liver are well represented by: Budd, *Diseases of the Liver*, 1845, 2d edition, 1853, 3d edition, 1857; Frerichs, *Klinik der Leberkrankheiten*, 1858-61 (English translation by Murchison, 1860-61); Bamberger, *Krankheiten der Leber*; Virchow's *Handbuch der speciellen Pathologie und Therapie*, vol. vi, 1855, 2te Aufl., 1864; Murchison, *Clinical Lectures on Diseases of the Liver*, 1868, 2d edition, 1877; and Ponfick, Thierfelder, Schüppel, Leichtenstern, and Heller, *Krankheiten der Leber*, von Ziemssen's *Handbuch der speciellen Pathologie und Therapie*, vol. viii, 1878 (English translation, 1880). Progress in the development of our knowledge is exemplified by: Harley, *Diseases of the Liver*, 1883; Waring, *Diseases of the Liver, Gall-bladder, and Biliary System*, 1897; Gilbert, Fournier, Garnier, and Surmont, *Maladies du foie*; Brouardel and Gilbert's *Traité de Médecine et de Thérapeutique*, 1898; Lancereaux, *Traité des maladies du foie et du pancreas*, 1899; Quinke and Hoppe-Seyler, *Krankheiten der Leber*; Nothnagel's *Handbuch der speciellen Pathologie und Therapie*, vol. xviii, 1899 (English translation, 1903); Chauffard, *Maladies du foie et des voies biliaires*; Charcot, Bouchard, and Brissaud's *Traité de Médecine*, vol. v, 1892, 2me edition, 1902; Hunter, White, Davidson, Lafleur, Hawkins, Robson, and Thomson, Allbutt's *System of Medicine*, 1900, vol. iv; Mongour, *Précis des maladies du foie et des voies biliaires*, 1905; and Weintraud, *Diseases of the Liver*; von Noorden's *Metabolism*, 1907, ii, 229. Unquestionably the best book on the subject is Rolleston's *Diseases of the Liver, Gall-bladder, and Bile Ducts*, 1905, a record of much personal observation, and a thorough review of the literature with many statistics. A good review of the literature will be found also by von Bardeleben, *Centralblatt für die Grenzgebiete der Medizin und Chirurgie*, 1906, ix, 722, 777, 817, 860, 894.

insidious onset and more chronic course, the time of onset cannot be determined, and frequently structural alterations are well advanced before obtrusive symptoms develop. It is difficult to believe, however, that normal liver cells can perform their functions otherwise than normally, and, conversely, abnormal function presupposes structural alterations—macroscopic, microscopic, or physicochemical. In some instances these structural alterations cannot be detected by the means of investigation at present at our command, but their existence cannot be denied, and we must look upon the sensible evidences of disease, the symptoms, as the manifestations of structural alterations already effected. As a rule, noteworthy symptoms are associated with more or less readily demonstrable structural alterations; but experience teaches us that extreme disorder of function is not infrequently associated with apparently normal structure, and that, on the contrary, in consequence of the power of adaptation, widespread and advanced structural alterations are not incompatible with long life and freedom from distress. It is essential, therefore, in disease as in health, to correlate function with structure, and to study diseased processes in the liver, as well as elsewhere, from the structural (or anatomical), the functional (or physiological), and the chemical points of view.

The functions of the liver may be said to be: (1) To detoxify poisons that enter the body by way of, or are elaborated within, the gastro-intestinal tract, or are perhaps produced elsewhere; (2) to secrete bile; (3) to warehouse some of the excess of fat taken as food and to release it when the supply from without becomes deficient; (4) to warehouse glycogen derived from the carbohydrates taken as food, or from the non-nitrogenous part of the proteins when the supply of carbohydrates is deficient, and to convert the glycogen into glucose and liberate it as it is required by the system; and (5) to assist in the metabolism of the proteins, to the extent, at least, of forming urea from ammonia compounds. There are other functions, such as erythrocytolysis, etc., that are of minor interest in the present connection.

Disorder of the functions of the liver may be primary or secondary (to structural alterations in the liver or to disease elsewhere); and it may be partial, involving perhaps only one function, or it may be total, involving presumably all the functions. The belief in a primary general disorder of function is well exemplified in the terms torpid liver, biliousness, bilious headache, lithæmia, etc., that have been handed down to us by our forefathers. Present-day opinions tend, perhaps unwisely, to minimize the influence of the liver in these disorders; but this seems an almost inevitable result of the knowledge that phenomena commonly attributed to a supposed torpid liver and the other affections mentioned may result from factors that do not involve the liver primarily, if at all, and that uric acid (lithæmia), by no means the important factor in disease it was once thought, is not formed exclusively in the liver. The dyspeptic symptoms of a supposed torpid liver are usually due to a gastro-intestinal catarrh set up by dietetic indiscretions, excessive eating (especially of proteins and carbohydrates), the consumption of too much alcohol, etc.; the spread of this catarrh to the diverticulum of Vater and the common bile duct, or perhaps rarely a toxic descending radicular cholangitis, is answerable for the subicteric tint of the conjunctiva and skin common in these subjects; and the headache and other nervous symptoms result from the absorption and circulation in the blood stream of enterogenic toxins. The liver usually participates in

the process only secondarily; perhaps the enterogenic toxins are produced in such amounts that the liver is incapable of neutralizing them and they pass over into the circulation, or they are so virulent that they impair the functional activity of or set up congestive and other alterations in the liver. In either event the symptoms are gastro-intestinal rather than hepatic in origin, and the liver, responding to excessive demands and being in consequence actively congested, is often, for a time at least, of increased rather than of decreased functional activity. The bearing of these facts on treatment is obvious and material.

There is, however, another series of cases, such as the passive congestion of chronic cardiac and pulmonary disease, in which there is reason to believe that the functional activity of the liver is reduced. It is usually difficult, if not impossible, in these cases to differentiate the symptoms properly referable to disordered functions of the liver from those really due to the defective circulation in general with consequent congestion of the different organs, notably the intestines and the kidneys, and defective elimination. It is not unlikely, however, that the headache and other nervous symptoms, digestive disturbances, and loss of flesh and strength that occur in these cases are due, in part at least, to the disordered functions of the liver. This is perhaps due directly to the influence of enterogenic poisons which, being carried to the liver by the portal circulation, encounter an organ reduced in vitality by the long-continued passive congestion, and incapable of exerting as it should its detoxifying function; the toxins, in consequence, pass over into the general circulation and cause at least some of the symptoms.

In another series of cases, more or less complete functional failure of the liver ensues. These are cases of severe intoxication or infection, in which widespread destruction or disorganization of the hepatic parenchyma occurs. The striking clinical phenomena, which may develop suddenly, although they are sometimes preceded by minor symptoms, are for the most part nervous; they are variously designated hepatargia, acholia, cholæmia, hepatic auto-intoxication, etc. Headache is a conspicuous symptom, and is soon followed by mental excitement, delirium (which may be very active or maniacal), muscular twitchings, and convulsions; or mental hebetude progressing to deep coma may ensue. Most of these cases are associated with varying grades of jaundice and other phenomena that will be more fully discussed under jaundice and acute yellow atrophy of the liver.

From time to time tests for determining the functional activity of the liver have been advanced, but few have withstood critical investigation, and we have made scarcely any progress in our endeavors to correlate definite disorder of function with definite alterations of structure. This is attributable, in part, to the complexity of the processes involved; but it is due also, in part at least, to the fact that the functions of the liver are not altered specifically by what we recognize grossly as definite diseased processes; that certain functional disturbances are not manifested by structural alterations susceptible of detection by the means at present at our command; that the healthy portion of the liver may act compensatorily for that diseased or destroyed; that other parts of the body have, or under the stress of circumstances may assume, functions akin to those of the liver, that is, act vicariously for the liver; that the secretion of the liver, the bile, can rarely be examined or studied directly; and that there is often difficulty in distinguishing between symptoms that may be attributed to hepatic insuffi-

ciency (hepatargia, acholia), to absorption of bile acids and bile pigments (chokemia), or to the initiating infection or intoxication.

The Detoxifying Function.—Certain substances highly poisonous when administered hypodermically are much less harmful or entirely innocuous when administered by the mouth. In some cases, perhaps, this detoxification may be brought about by the gastric and the intestinal secretions; but in many cases the poisons are carried by the portal circulation to the liver, and there, probably by a process of oxidation, deprived of their toxicity. This obtains in the case of substances such as curare, nicotine, etc., and to a less extent in the case of substances such as morphine, strychnine, etc. The liver is believed to exert a similar detoxifying action on the toxins of certain bacteria, such as the typhoid bacillus, etc., and on the products of bacterial putrefaction in the intestine, such as indol and phenol. Doubtless in some cases these poisons are excessive in amount or in virulence, and the liver is unable successfully to cope with them. Disturbances in this protective or detoxifying function of the liver are believed by some observers to be the basis of the eclamptic manifestations of uræmia, pregnancy, etc.; but the question still awaits definite solution.

This detoxifying function of the liver was early studied by Bouchard, Roger, and others of the French school. Believing that this function should be notably interfered with in disease of the liver, they postulated the theory that the non-neutralized toxins should appear in the urine, and that a study of the urinary toxicity should prove a serviceable clinical diagnostic test. Unfortunately, the results of their experiments on lower animals have not received confirmation by later investigators, and the question, although energetically pursued and combated by the French school, seems to have been definitely settled by Gueirolo, who found no abnormal toxicity of the urine of dogs in which an Eck fistula had been made. Schapiro believed that in disease of the liver strychnine appeared in the urine more rapidly than normally, and advanced this as a test of the functional activity of the liver. Other substances have been similarly studied, such as antipyrine by Capitan and Gley, and cocaine by Gley and du Val, but these tests have not withstood critical study. There is no doubt that in disease the detoxifying function of the liver is more or less impaired, but there is no trustworthy method of determining and utilizing this as a diagnostic test clinically. The results of some recent studies by Hawk,¹ however, suggest the wisdom of restricting the amount of meat in the diet in autotoxic conditions, since the use of meat appeared to increase the toxic phenomena.

The Lipogenic Function.—In health the warehousing of fat in the liver is a function of less importance in man than in some of the lower animals, especially those that have little power to accumulate fat in the muscles and other tissues. In disease, however, the liver may store considerable fat, but only when the amount of stored glycogen becomes much decreased or disappears. Disturbances of fat metabolism may be attended by extremely serious consequences, notably the condition spoken of as acidosis, due to the presence of the acetone bodies—acetone, diacetic acid, and β -oxybutyric acid. These substances are formed principally in the liver and the muscles, but probably also in other tissues. Although formed from the fats of the body in certain conditions when the oxidizing powers are reduced,

¹ *American Journal of Physiology*, 1908, xxi, 259.

they are directly related to deficiency of glycogen—which may be the consequence of insufficient intake (as in starvation) or of excessive output (as in diabetes). In certain chronic diseases of the liver, such as cirrhosis, in which the alkalinity of the blood and therefore of the pericellular fluids in the liver may be reduced, an additional acidosis due to disturbed or perverted fat metabolism, may lead to widespread dissolution of the hepatic cells, to autolysis the consequence of activity of proteolytic ferments. The attendant phenomena are much akin to those of acute yellow atrophy of the liver.

The Urea-forming Function.—The synthesis of urea from ammonia compounds and the availability of disturbances in this function as a test of hepatic insufficiency have also been much studied. A priori it is not unreasonable to suppose that in case the activity of the liver be reduced the amount of urea in the urine should be diminished and that of ammonia increased. This opinion has received some support from the experimental studies, by means of Eck fistulæ on dogs, of Hahn, Massen, Nencki, Pawlow, and Salaskin. In jaundice from obstruction the output of urea is usually not affected, although the percentage of ammonia is increased. In cirrhosis, on the other hand, the urea is diminished and the ammonia is much increased—which lends support to the theory of disturbed urea synthesis; but this theory is more or less discredited by the fact that feeding ammonia salts to cirrhotic subjects is not followed by impairment of the formation of urea. Stadelmann advanced the theory of acidosis, seeking to explain the increased ammonia output on the purely chemical basis of the neutralization of an excess of acid (as is seen in diabetes). The volatile fatty acids have been found increased (von Jaksch, Stadelmann, von Noorden, Weintraud); sarcolactic acid (Schultzen, Riess, Fraenkel, etc.) and diacetic acid (Senator and Soetbeer) have also been found—which, in connection with lessening of the alkalinity of the blood and increase of bases in the urine (Münzer and Soetbeer), tends to confirm the theory of acid intoxication. Therapeutically the opinion has received support from Münzer, who found a decrease of the ammonia after the use of sodium bicarbonate. There is, however, this serious objection to using the amount of urea as an indication of hepatic insufficiency: urea is formed in the body elsewhere than in the liver. The ammonia output also is not a trustworthy test, since every acidosis—whatever its origin—is associated with an increase of ammonia.

The Glycogenic Function.—This has been much studied with a view to discovering, if possible, departures from the normal and utilizing these in clinical diagnosis. Stoppage of the flow of bile is supposed to prevent the storing of glycogen. Hergenhahn, after ligating the bile ducts, found only extremely small amounts of glycogen in the liver, despite various feedings; even injections of cane sugar did not cause glycosuria—which leads to the supposition that the liver is not necessarily deprived of its glycogen-forming function, but rather has trouble in warehousing glycogen, and that a hepatic patient should readily exhibit alimentary glycosuria. Glycosuria, as a matter of fact, has been observed in certain disorders of the liver. Gans claims to have found it in many subjects during attacks of so-called colic, and Exner states that of forty such, only one failed to exhibit glycosuria. The obvious inference has been combated by Naunyn, Kausch, and others. The weight of opinion is that alimentary glycosuria is not a trustworthy test of hepatic insufficiency.

A new impetus to the study of alimentary mellituria was given by Sachs, who found that dehepatised dogs showed a lessened tolerance for levulose, but not for dextrose, galactose, and arabinose. Studying the subject clinically, he found that healthy as well as diabetic subjects have a greater tolerance for levulose than for dextrose, and that the subjects of hepatic disease have a lessened tolerance for a like amount of levulose. Strauss, applying these results in a large series of cases, found that 90 per cent. of hepatic subjects presented levulosuria after the ingestion of a measured amount of levulose. Subsequently his observations were confirmed by Baylac and Armand, Raspide, Bruining, Ferannini, Steinhaus, Lépine, Crisafi, Samberger, Chajes, and von Halász. Alimentary levulosuria thus has been suggested and utilized as a test of hepatic insufficiency. Although its trustworthiness has been questioned by a few observers, such as Landsberg, the weight of opinion is that it is of distinct value. It is much more satisfactory than alimentary glycosuria. Sachs found that levulose is converted into glycogen only in the liver; that the muscles do not possess this power. Glucose, as is well known, is acted upon by the muscles, and its conversion into glycogen is not necessarily limited to the liver; Külz has determined that when the glycogenic function of the liver is impaired the muscles act compensatorily. The reasons for this difference in the metabolism of glucose and of levulose have not been determined.

Of the many disorders of the liver, alimentary levulosuria seems to be of special diagnostic significance in cirrhosis. It has not been found in many non-hepatic diseases; pneumonic subjects are said to exhibit both spontaneous as well as alimentary levulosuria, but these are not likely to be confused with the subject of diseases of the liver.

The test should be performed as follows: Food should be withheld after the evening meal until the test has been completed. In the morning the patient should void his urine, and then (upon an empty stomach) he should take 100 grams of levulose dissolved in 500 cc. of water. Weak tea without cream may be substituted for the water if preferred by the patient. At the end of an hour, and hourly thereafter for four hours, the patient should void his urine. The specimens should be examined separately for levulose, preferably by the polariscope, or by the Seliwanoff method, which is as follows: Equal parts of the urine and of a half-and-half solution of concentrated hydrochloric acid and water should be mixed in a test-tube, and a few grains of resorcin added. The mixture should then be boiled, whereupon, if levulose is present, a beautiful red color appears; this deepens on standing and a dark-red deposit settles in the test tube. If the disorder of the liver be of moderate severity the levulose will be found in the third or fourth specimen of urine, perhaps in the fourth only; if there be profound alteration of the hepatic structure the levulose may be found in the first specimen of urine, as well as in the subsequent specimens, and it may persist for several additional hours. Urine when boiled with hydrochloric acid often gives a pinkish color, but this is transient and soon fades, and is not likely to cause confusion. The test rarely gives rise to subjective symptoms; occasionally a mild diarrhoea ensues. Should the disorder of the liver be associated with diarrhoea the test may fail on account of rapid peristalsis preventing proper absorption of the levulose.

JAUNDICE.

Jaundice is a term applied to staining of the tissues with bile pigments—bilirubin and biliverdin. It is recognized clinically by a yellowish or yellowish-green discoloration of the skin and visible mucous membranes and by the presence of bile pigments in the urine. It is not a disease *sui generis*, but merely a symptom of a wide variety of disorders. Although it indicates some disturbance in the secretion or excretion of bile, and is therefore significant of disease of the liver, it occurs also in disorders in which the liver is not involved primarily.

Jaundice occurs in a variety of circumstances. In many cases it results from an obstruction to the free flow of bile. Often the obstruction is obvious and consists of: (1) An obstruction within the common bile duct or the hepatic duct, such as gallstones, parasites, foreign bodies, etc.; (2) inflammatory, cicatricial, or neoplastic stenosis of the ducts; (3) compression of the ducts from without by tumors of the pancreas, stomach, intestine, gall-bladder, regional lymph glands, kidney, retroperitoneum, mesentery, etc., inflammatory adhesions, swollen glands, faecal accumulations, aneurism of the aorta or of the hepatic or mesenteric artery, etc.; or (4) kinking or torsion of the ducts the consequence of gastropptosis, hepaptosis, nephropptosis, the pregnant uterus, tumors of the abdominal or pelvic organs, etc. In another series of cases—of disease of the liver unattended by gross obstruction of the extrahepatic biliary ducts—the hindrance to the free flow of bile is none the less demonstrable, if less marked and less obvious at first sight; these comprise cases of localized inflammatory processes (abscesses), tubercles, gummas, hydatid cysts, primary and secondary new-growths within the substance of the liver, etc. Jaundice, however, occurs under other circumstances—in which apparently the biliary ducts are patent, as in cirrhosis and other diffuse diseases of the liver; in many infections, such as the different types of so-called infectious jaundice, syphilis, yellow fever, septicopyæmia, malaria, pneumonia, typhoid fever, etc.; in intoxications, such as poisoning with ptomaines, phosphorus, arseniuretted hydrogen, chloroform, mushrooms, toluylenediamin, pyrogallol, snake venom, coal-tar products, etc.; in acute yellow atrophy of the liver; in progressive pernicious anæmia and hæmoglobinæmia; in disturbances of the circulation, such as passive congestion; in certain nervous perturbations (so-called emotional jaundice, menstrual jaundice, etc.); in the newborn, etc.

Types of Jaundice.—The aforementioned circumstances in which jaundice occurs have led to different classifications of the disorder—variously based upon the supposed pathogenesis, the etiology, the clinical features or course, etc. It must be apparent that jaundice can result only from: (1) An obstruction somewhere in the course of the biliary tract in consequence of which the bile becomes absorbed by the lymphatics or the bloodvessels—jaundice from stasis of bile; or (2) disturbances in the functions of the liver cells, whereby the bile is diverted from the biliary capillaries to the lymphatics or the blood channels—jaundice from parapptosis of bile. In the one case the disorder involves the bile after it has been formed and entered the biliary channels; in the other it involves the bile pigments while they are still within the hepatic cells. There is comparatively little discussion regarding the nature of the cases due to obvious obstruction of the extrahepatic or

the intrahepatic ducts; these are commonly described as obstructive or mechanical jaundice. Regarding the cases of so-called non-mechanical or non-obstructive jaundice there are different opinions. A mistaken interpretation of surface phenomena led to a classification into: (1) Hepatogenous jaundice, that in which the liver, including the biliary ducts, was obviously at fault, that is, obstructed; and (2) hæmatogenous jaundice, that in which no disorder of the liver or the biliary ducts being apparent, the bile pigments were supposed to be formed in the bloodvessels in consequence of the destruction of erythrocytes and the conversion of the thus liberated hæmoglobin into bilirubin. The work of Naunyn, Minkowski, Stadelmann, and others, however, effectually proved that all jaundice is hepatogenous—in the sense that bile pigments are formed only in the liver; these pigments are not formed when the liver has been removed or the afferent vessels ligated. Nevertheless, the influence of increased destruction of erythrocytes in providing the material (hæmoglobin) from which bile pigments are formed in excess (polychromia) in some cases of jaundice cannot be gainsaid; this has led to the use of the term hæmo-hepatogenous jaundice. Since this form of jaundice is found in toxic, infectious, and other conditions associated with destruction of erythrocytes, it is spoken of as toxic, infectious, or hæmolytic.

Inasmuch as the etiological factor in many of the cases of so-called non-obstructive jaundice is unknown, it is quite impossible to differentiate them clinically or otherwise, aside from the severity of the symptoms; but the clinical designations, mild and severe, are often quite inappropriate, since the degree of the jaundice and the severity of the other symptoms frequently bear no relationship whatever the one to the other. There can be little question, however, that many of the cases represent only different stages or grades of the one process. In a simple and usually mild form, jaundice is an accompaniment or sequel of gastro-intestinal catarrh set up by dietetic or other indiscretions or by certain toxic or infectious processes; the direct cause of the jaundice is an extension of the catarrhal process to the diverticulum of Vater or the common bile duct and consequent obstruction to the flow of bile. Jaundice is often the only noteworthy symptom, other symptoms being relatively or actually in abeyance. Since these cases commonly pursue a benign course, and the few that have come to necropsy have revealed only catarrhal papillitis or cholangitis, the disorder is spoken of as simple or catarrhal jaundice (preferably catarrhal cholangitis). In another series of cases in addition to the jaundice there are phenomena of general infection, such as fever, neuromuscular pains, enlargement of the spleen and liver, gastro-intestinal disorders, such as vomiting and diarrhœa, nervous manifestations, such as headache, stupor, etc., often leukocytosis, albuminuria, tube casts, etc.; to these cases the term infectious jaundice (Weil's disease) has been applied.¹ The not uncommon epidemic occurrence of such cases warrants the use of the term epidemic jaundice. In other cases the clinical phenomena are more severe, symptoms attributable to hepatic insufficiency (hepatargia), to poisoning by bile acids (cholæmia), or to hepatic auto-intoxication (acidosis), supervene, and a fatal issue usually ensues; the condition is spoken of as grave jaundice (*icterus gravis*). When, in addition, considerable destruction of the liver cells occurs, the term acute yellow atrophy of the liver is

¹ Consult Boggs in vol. iii, p. 528, of this work

employed. No sharp line of distinction can be drawn between these cases; cases mild in the beginning (seemingly catarrhal jaundice) may progress to the most severe and fatal (acute yellow atrophy); the degree of jaundice bears no constant relationship to the demonstrable changes in the liver, nor, as already stated, to the severity of the other clinical phenomena; in some cases of unusual and widespread destruction of the hepatic parenchyma the jaundice may be slight, rarely even entirely absent—so that, until such time as the etiological factors shall have been better elucidated, we perhaps must rest content with the terms toxic, infectious, hæmolytic, or hæmo-hepatogenous jaundice, as seems best suited to specific cases. Some of the cases are unquestionably examples of typhoid infection of the biliary tract others are likely due to infection with *Bacillus coli communis*, *Bacillus proteus*, etc.; perhaps others are due to infection by unidentified (anaërobic?) bacteria which, settling in the duodenum, multiply and produce an ascending cholangitis and ultimately more or less destruction of the hepatic cells. Other cases, toxic in nature, result from the action of ptomains and other poisons transported to the liver by way of the portal circulation. In still other cases the primary action of the poison is on the erythrocytes (hæmolysis), in consequence of which an increased amount of free hæmoglobin is carried to the liver cells and transformed into bile pigments.

Pathogenesis of Jaundice.—The mechanism of jaundice due to gross obstruction of the extrahepatic biliary ducts, or to none the less real if less marked obstruction of the larger intrahepatic ducts, is readily comprehended. On the one hand, the outflow of bile into the duodenum is hindered; on the other hand, as has been conclusively proved, the hepatic cells, even after the common bile duct has been completely occluded, continue to secrete bile (although perhaps in lessened amount than normally), to convert into bile pigments the free hæmoglobin brought thither. In consequence, then, of obstructed outflow and of continued production, bile accumulates within the biliary tract, the pressure increases, and the biliary ducts become dilated. It has been demonstrated by Eppinger¹ that this dilatation involves not only the larger biliary ducts, but extends to the finest biliary capillaries, which become enormously distended, especially at the points of communication of the capillaries of adjacent rows of liver cells. Furthermore, evidences of the increased pressure are seen in the intercellular biliary capillaries, which become lengthened in the direction of their distal end, dilate ampulla-like, and gradually approach and finally rupture into the lymph spaces between the hepatic cells and the bloodvessels; thence, by way of the thoracic duct, the biliary constituents reach the bloodvessels. That the pathway of the bile to the bloodvessels is by way of the lymphatics and the thoracic duct has been repeatedly demonstrated—by Saunders (1809), and later by Fleischl, Kufferaht, Harley, Frey, and others: when the common bile duct is ligated bile pigments may be recovered soon from a thoracic-duct fistula, but not from the blood, and if the common bile duct and the thoracic duct be ligated, biliary pigments do not appear in the urine for many days.

The mechanism of jaundice not due to obvious or gross obstruction of the biliary ducts is not so well understood; it has been variously explained. Frerichs believed that the bile found its way directly into the bloodvessels

¹ *Ergebnisse der inneren Medizin und Kinderheilkunde*, 1908, i, 107 (literature).

in consequence of changes in the tension of the contents of the hepatic cells and of the bloodvessels—either an increase in the intercellular pressure or a decrease in the portal pressure. Liebermeister believed that the disordered liver cells lost their ability to retain or fix the bile, and that in consequence it became diffused into the bloodvessels or lymphatics—so-called diffusion or akathetkic jaundice. Minkowski assumed a disturbance in the normal currents within the liver cells, so that the bile became diverted from the biliary capillaries to the bloodvessels or lymphatics—jaundice from parapetesis (paracholia of Pick). It is by no means unlikely, indeed, it is quite probable, that the toxic and infectious causes of this form of jaundice seriously compromise the functional integrity of the liver cells; but it has also been demonstrated, experimentally and otherwise, that they provoke also a radicular cholangitis—which obstructs the free flow of bile. Eppinger states that in these cases thrombi made up of coagulated bile occur in and obstruct the biliary capillaries; in consequence thereof, mechanical stasis of the bile develops in the finest biliary capillaries, and, as in cases of gross obstruction of the larger extrahepatic ducts, the biliary canaliculi dilate and ultimately rupture into the pericellular lymph spaces. The bile thrombi are interpreted as a manifestation of biliary inspissation, perhaps related to coagulation of a pathological albuminous exudate (to which the liver cells become pervious); but the basic cause is believed to be marked erythrocytolysis in consequence of which the liver is forced to the production of an increased amount of bile pigments. Experimentally, it has been found that in these cases there is at first an increased formation and excretion of bile (polycholia) and of bile pigments (polychromia); soon, however, the excretion of bile lessens, the bile that is excreted becomes viscid, and finally excretion may cease—which may be attributed to a radicular cholangitis or the biliary thrombi, or both. Thus although this form of jaundice is properly called toxic, infectious, or hæmolytic, it is at the same time hepatogenous and obstructive. The degree of jaundice is determined by the degree and extent of the obstruction rather than by the amount of hæmolysis: a high grade of jaundice may occur with comparatively little hæmolysis, whereas considerable hæmolysis may be attended with only slight jaundice.

Icterus neonatorum is a peculiar form of jaundice that develops in from two-thirds to three-fourths of newborn infants on the second or third day of life, and usually fades within a week or ten days. The cutaneous discoloration is slight, and is commonly unattended by other symptoms. The urine is often of normal color, and usually does not contain soluble bile pigments, but may reveal biliary granules or crystals in desquamated renal epithelium. The nature of this form of jaundice is not well understood. It has been attributed to compression of the biliary capillaries by distended portal radicles; to catarrhal cholangitis (Virchow); and to biliary stasis in consequence of the diminutive size of the bile ducts and consequent obstruction (Kehrer and Cohnheim). Frank attributed it to absorption of bilirubin from the meconium; and Quincke has suggested that this bilirubin, which is not converted into urobilin on account of the absence of intestinal fermentation in the newborn, being absorbed, reaches the general circulation by way of the ductus venosus without traversing the liver. Perhaps the disorder is really due to the erythrocytolysis of the early days of life. If this be the true explanation, the pathogenesis is that of hæmolytic jaundice, already discussed. This benign form of jaundice must be dis-

tinguished from a more severe and often fatal form that may also occur in the early days of life in consequence of congenital syphilis, septicopyæmia, congenital obliteration of the bile ducts, etc.

A so-called *nervous* or *emotional jaundice* has been occasionally commented upon. It is said to be provoked by sudden emotional disturbances, such as fright, shock, anger, etc., and has been attributed to psychically induced spasm of the unstriped muscular fibers about the insertion of the common bile duct into the duodenum with consequent stasis of bile, and to lowering of the portal pressure with consecutive reversal of the biliary circulation within the hepatic cells. The cases are largely from the older literature, and were observed at a time when the nature and etiology of jaundice were less well understood than at present; they must be accepted with the greatest reservation. Equally subject to doubt are cases of so-called *menstrual jaundice*.¹ Disturbances of menstruation are so common and associated jaundice so rare as to lead to the conclusion that the one are in no way related to the other, that their occurrence together is purely accidental, and that the jaundice is due to some one of the more likely causes; gallstones have been found in a case of supposed menstrual jaundice.

Finally, a so-called *hereditary* or *family form of jaundice* has been described. The cases are very rare. Undoubtedly all are not of one nature; some are examples of ordinary icterus neonatorum, others of congenital obliteration of the bile ducts, others of infections not uncommon in the early days of life, others are related to enlargement of the liver and spleen (types of hypertrophic cirrhosis of the liver), whereas others are perhaps of undetermined nature.

Effects of Jaundice.—The clinical phenomena attendant upon jaundice are due largely to the underlying causes of which the jaundice itself is merely a symptom. The symptoms, however, inasmuch as they are related directly to the jaundice and not to the underlying cause, are due, on the one hand, to the presence of biliary derivatives in the circulating blood, and, on the other hand, to the absence of bile from the intestine. All of the tissues and organs, with the exception of the nervous, are stained with biliary pigments; and in some severe cases of toxic or infectious jaundice the nervous system also may be stained. The staining is due, in part, to the deposition of biliary pigments in the tissues, in part to the circulation of blood and lymph containing undeposited pigments; it is most marked in the skin, mucous membranes, serous membranes (endocardium, intima of vessels, etc.), the liver, kidneys, heart, etc., serous transudates, exudates, etc.

The Mucous Membranes and the Skin.—Jaundice is manifested first by a yellowish discoloration of the conjunctivæ; in mild cases the discoloration is demonstrable there only. In the other visible mucous membranes, except that of the hard palate, the jaundice, unless of high grade, becomes apparent only after the tissues, by pressure, have been deprived of blood; the mucosa of the hard palate, however, being normally pale may reveal the discoloration without this.

The skin becomes of a characteristic lemon-yellow, orange-yellow, greenish-yellow, olive, or olive-bronze color. The discoloration is light in many cases of toxic or hæmolytic jaundice, in which it is rarely more than moderate in grade; it is light also in mild cases of catarrhal cholangitis; it is darkest in cases

¹ Consult Metzger, *Zeitschrift für klinische Medizin*, 1904, liii, 149 (literature).

of permanent and complete obstruction of the common bile duct, and it may become so dark—olive bronze or greenish black—as to warrant the designation black jaundice (*melanicterus*, *icterus melas*). The intensity of the jaundice in these cases is proportionate to the completeness of the obstruction of the common duct, or, in case the obstruction involve one of the larger intrahepatic ducts, to the size of the duct implicated. The jaundice occurs earliest (sometimes within several hours) in cases of obstruction to the finer biliary ducts and capillaries, on account of the seat of the obstruction; it is delayed, commonly four or five days, in obstruction of the common duct, because the bile accumulating gradually distends the larger ducts and the gall-bladder; time is required to produce sufficient pressure in the biliary canaliculi to cause their rupture into the adjacent lymphatic spaces and consequent absorption of bile. The pigment for the most part is deposited in the cells of the rete Malpighii, where in great measure it persists, often causing an obvious discoloration until the cells have been desquamated. It is for this reason that the cutaneous discoloration commonly persists a week, perhaps several weeks, after bile pigments have disappeared from the urine.

Of other cutaneous manifestations, pruritus is the most common and distressing. Rarely it precedes the discoloration of the skin;¹ it is likely to develop in cases of jaundice of sudden onset; as a rule, it is most marked in protracted and severe cases; but it bears no definite relationship to the duration or the severity of the jaundice. The cause of the pruritus is not known; it is apparently not due to the deposited bile pigment; perhaps it is due to an associated toxæmia that varies in degree, as does the pruritus, from time to time independently of the jaundice. Urticaria, perhaps due to a toxæmia, is not uncommon. Considerable sweating, often localized to the palms, etc., is sometimes observed. Local infections, such as boils, occasionally occur. In chronic cases, local telangiectases of the skin (sometimes also of the oral and other mucous membranes) tend to develop, and flat or nodular yellowish, chamois-leather-colored patches (*xanthelasma*, *xanthoma*, *vitiligoidea*²) may appear on the skin or mucous membranes. These are most common in the skin of the eyelids, and in the folds and creases of the joints; they tend to be symmetrical in distribution; they have been observed on the gums, in the mucosa of the biliary ducts (perhaps in part answerable for the jaundice), etc. They are believed to be inflammatory or endothelial in origin, and to be due to the jaundice or an associated toxæmia.

The Blood.—Bile pigments are present in excess in the circulating blood, but whether they exert any deleterious effect on the blood itself has not been satisfactorily determined. Bouchard and de Bruin claim to have found lessened toxicity of the bile after removal of the pigments, and that a small dose of pure pigments suffices to kill experimental animals—probably by an action on the heart, kidneys, and central nervous system; but their work has been adversely criticised, and the question is still in doubt. The probable slight toxicity of the bile pigments is perhaps answerable for the lowering of the bodily temperature. The major toxic action of the bile is due to the bile acids—salts of glycocholic and taurocholic acids. Glycocol and taurin result from hydrolysis of protein; the source of cholic acid is not definitely

¹ Consult Riesman, *American Medicine*, 1907, xiii, 77 (literature).

² Consult Futeher, *American Journal of the Medical Sciences*, 1905, cxxx, 939.

known. At the inception of jaundice these acid salts may be found in the circulating blood, but after the jaundice has become well established they usually disappear from the blood, being apparently produced in lessened amount, although the bile pigments continue to be produced in normal or almost normal amount. The toxic action of the bile acids is exerted on the blood, the cardiovascular system, the kidneys, the central nervous system, the liver, the muscles, etc. In ordinary obstructive jaundice, at least when it is well developed, the amount of bile acids in the blood is not sufficient to produce any noteworthy hæmolysis; indeed, polycythæmia is not uncommon. In toxic or infectious jaundice, however, hæmolysis is common; usually, it is itself the direct cause of the jaundice, but it may be increased by circulating bile acids. Chauffard¹ believes in the possibility of differentiating hæmolytic from ordinary obstructive jaundice by the fragility, granular appearance, and small size of the erythrocytes (those from 3 to 6 μ in diameter increasing to 15 to 18 per cent. of the total number); in obstructive jaundice the erythrocytes are said to be normally resistant, of normal size, and free from granulations. The clotting time of the blood is much increased in jaundice—from the normal three to four minutes to ten to twelve minutes, a fact of much importance when surgical operations in jaundiced patients are under consideration. In protracted and severe cases there is a marked tendency to subcutaneous, submucous, and subserous hemorrhages. The alkalinity of the blood is but slightly changed, if at all, except perhaps in some cases of cirrhosis with jaundice in which it may be somewhat reduced. The dried weight and the specific gravity are unaltered. The not uncommon property of the blood serum of jaundiced patients to agglutinate typhoid bacilli is probably due, not so much to the jaundice per se, as to the fact that the jaundice is a manifestation of typhoid infection.

The Heart and Bloodvessels.—Bradycardia is a characteristic feature of jaundice, especially of acute cases and those of short duration. The pulse rate may fall to 60, to 40, even to 21 (Frerichs) a minute, and it is subject to sudden fluctuations upon slight provocation. This has been attributed to a local inhibitory action of the bile acids on the cardiac muscle or ganglia; but recent investigations suggest that perhaps the action is on the central nervous system. In chronic cases the bile acids become much reduced in amount or disappear, or the cardiac muscle becomes accustomed to their influence, and the bradycardia usually lessens or disappears. In cases of long duration a weak and diffuse apex beat, increase in the area of cardiac dullness, a systolic murmur at the apex, an accentuated second pulmonic sound, low blood pressure, and other evidences of myocardial insufficiency are not uncommon. The respirations also may be reduced to ten or less a minute.

The Urine and Other Excretions.—In jaundice a considerable portion of the biliary derivatives is excreted in the urine, which, in consequence, is discolored yellowish, yellowish red, greenish, or greenish brown, depending upon the relative amounts of bilirubin, biliverdin (its oxidation product), or urobilin (its reduction product). These bile pigments may be found in the urine several hours, sometimes even several days, before there is any obvious discoloration of the skin or visible mucous membranes; they constitute thus the first clinical manifestation of jaundice. A yellowish or yellowish-green tint to the froth, upon shaking the urine, is quite character-

¹ *Semaine médicale*, 1908, xxviii, 49.

istic; and this or a similar stain given to filter paper usually suffices for the recognition of icteric urine but recourse may be had to Gmelin's test. In addition to the bile pigments, the urine often contains a number of hyaline, sometimes bile-stained, casts—attributable to irritation of the convoluted and Henle's tubules provoked by excreting the bile pigments (which are not excreted by the glomeruli). In other and particularly long-standing cases, in addition to casts, the urine is diminished in amount, of increased specific gravity, and contains more or less albumin—an expression of nephritis due probably to associated toxic conditions rather than to the jaundice per se. The total nitrogen and the urea excretion are but slightly modified, but the ammonia excretion is usually increased. An increased excretion of uric acid also has sometimes been observed, but it is usually due to the quality of the food. The fatty acids and the neutral sulphur are said to be increased, the latter in consequence of diminished oxidation in the liver, but since disorders other than hepatic manifest a similar condition, this cannot be regarded as significant of insufficiency of the liver. The behavior of the amino acids in disease of the liver is still under investigation; sufficient work has not yet been done to permit one to form a just estimate of their diagnostic significance.

Urobilinuria¹ is commonly observed in disease of the liver. Urobilin as well as urobilinogen are formed in the intestine by the action of bacteria on bilirubin; they are absorbed and carried to the liver by the portal circulation. They are found in the urine especially when the liver is disordered, and in chronic disease of the liver they are believed by Edsall² to be significant of cirrhosis when there is no passive congestion, obstruction of the biliary ducts, or carcinoma. Fischler,³ also, recently has directed attention to the diagnostic significance of urobilinuria and urobilinogenuria, stating that practically they are found only when the liver is diseased, as in cirrhosis, passive congestion, etc., and that when found in infections, tuberculosis, etc., they are evidence also of disordered liver. The question needs further elucidation. The occurrence of urobilinuria in cases of jaundice, however, does not justify the designation "urobilin jaundice," since the cutaneous pigmentation is due to bilirubin and biliverdin.

Edsall also suggests that the presence of much phenol in the urine may be of some diagnostic significance in disease of the liver. Indicanuria is of no diagnostic significance whatever. As a matter of fact, the various urinary tests of hepatic insufficiency suggested from time to time are of very little practical importance; they scarcely even suggest disorder of the liver before its presence is unmistakable by the development of other clinical manifestations.

Excretions other than the urine also are stained in jaundice, such as the sweat (staining of the personal and bed linen), serous exudates and transudates, etc. The saliva, tears, mucus, and milk are not stained, unless a pathological inflammatory exudate be added to them. The sputum of complicating croupous pneumonia is stained, but not that of bronchitis.

The Liver.—Although it is quite impossible to study the bile during life, except in those cases in which biliary fistulæ have been made, it is probable

¹ Consult Hildebrant, *Zeitschrift für klinische Medizin*, 1906, lix, 351.

² *University of Pennsylvania Medical Bulletin*, 1904, xvi, 427.

³ *Münchener medicinische Wochenschrift*, 1908, lv, 1421.

that during the persistence of the biliary obstruction bile is formed in lessened amount (oligochohia, hypochohia). In some cases bile pigments appear to be formed in normal amount; in some cases they seem to be lessened. French authors have described a colorless bile, which is said to contain bile acids and cholesterin. The lessened amount of bile acids (in the blood, urine, and fæces) has been explained upon the assumption that, being formed in normal amount, they are in part destroyed in the blood; but Stadelmann has shown that bile acids injected intravenously are excreted almost quantitatively in the bile, and Nissen, Loewenton, Winteler, and Goodman¹ have shown that this is also true when the bile acids are administered by the mouth. Stadelmann has found experimentally that the production of bile acids is lessened when the liver cells have been poisoned; this is likely the cause in many cases of jaundice. The other functions of the liver also may be disturbed, reduction in the glycogen content and lessening of the detoxifying function. The liver cells sometimes are much degenerated, not always, however, directly in consequence of the jaundice. Eppinger has shown that in these cases marked regenerative changes occur to replace cells destroyed. In advanced cases of obstructive jaundice pericholangitic fibrosis sometimes occurs, and has been described as a form of biliary cirrhosis.

Nervous System.—Grave disturbances of the nervous system occur in many cases of jaundice; on the one hand, there are manifestations of depression, such as general neuromuscular weakness, asthenia, headache, vertigo, mental depression (that may progress to melancholia), insomnia, etc.; on the other hand, there are manifestations of irritation, such as severe headache, active delirium, coma, convulsions, etc. These manifestations are more common in cases of acute toxic or infectious jaundice (associated with fever) than in the more protracted cases of obstructive jaundice; but they may supervene in any case of jaundice and not uncommonly lead to the fatal issue. They are commonly described under the name cholæmia, but it is difficult to separate from one another the symptoms due to bile-acidemia, to interference with the detoxifying function of the liver, and to the initiating toxic or infectious process. The condition is not cholæmia in a restricted sense, that is, it is not due to bile-acidemia, since the symptoms may occur when bile acids are not in excess in the blood, as well as in cases of disease of the liver (such as cirrhosis) unassociated with jaundice. The condition is most likely an acid intoxication, due to disturbances of the detoxifying function of the liver, which may be brought about by disorganization of the hepatic parenchyma, such as occurs in severe forms of toxic and infectious jaundice, acute yellow atrophy of the liver, phosphorus poisoning, Eck's fistula, starvation, diabetes, etc., or by an excessive formation of enterogenic toxins, the consequence of absence of bile from the intestine. Xanthopsia and hemeralopia also are probably due to a toxic disturbance of the retina, rather than to the sometimes assumed discoloration of the media of the eye; were they due to the jaundice they should be much more frequent than they are, as well as proportional to the degree of the jaundice.

Gastro-intestinal Tract.—The absence of bile from the intestinal tract results, as a rule, in the passage of large, pale, grayish, slate-colored or clay-

¹ Hofmeister's *Beiträge zur chemischen Physiologie und Pathologie*, 1907, ix, 91.

colored, pasty, fœtid, acid stools. The pale color is due, in part only, to the absence of bile; in part it is due to the large amount of imperfectly digested fats, which may be increased from the normal 7 to 10 per cent. to as much as 80 per cent. Microscopically and chemically the fats are found to consist of free fatty-acid crystals, and soaps (calcium and magnesium) of the higher fatty acids; neutral fats are present in very small amounts. Rarely pale or decolorized stools contain biliary derivatives in considerable amount—determinable by chemical examination; this occurs especially in disease of the pancreas attended by obstruction to the outflow of the pancreatic juice but unassociated with jaundice. The pale color of the fæces in these cases is due to the large amount of fat,¹ and to the reduction of bilirubin to leuko-urobilin.² It is not always warrantable, therefore, to base on the color of the stools an opinion of the amount of bile that enters the intestine or of the degree of biliary stasis. The offensive odor of the stools is usually ascribed to the absence of bile which when present limits fermentation of the intestinal contents. Often, however, there is very little fermentation; Strasburger has demonstrated no undue protein decomposition; and the odor may be described as peculiar rather than offensive, being due to the higher fatty acids (Schmidt and Albu). Constipation is perhaps the rule, and is due to defective motility, the result of lack of the stimulating influence of the bile. Sometimes the bowel movements are frequent, perhaps diarrhœic, which may be due to large fæcal masses resulting from imperfect digestion of fat and to the laxative action of the free fatty acids. There is no effect on carbohydrate metabolism and very little on protein metabolism. Gastric hyperacidity is the rule, the biliary obstruction apparently causing an increase in the secretory activity of the stomach. Cases of associated gastric catarrh usually reveal hyperchlorhydria.

General Nutrition.—Man is able to withstand the effects of jaundice for a long time. The fatalities that ensue in the acute as well as in the chronic cases are due not so much to the jaundice per se as to other causes—the underlying cause of the jaundice, acid intoxication, etc. In long-standing cases, however, more or less disturbance of the general nutrition gradually ensues—due largely to imperfect fat absorption. To supply sufficient calories to prevent breaking down of the body tissues, there must be considerable increase in the protein in-take; this results in loss of appetite, disordered digestion, perhaps diarrhœa, etc., so that, the in-take being reduced, the patient must utilize his own fat or protein tissue to cover the continued loss, and, having no means of restoring the deficit, a very dangerous inanition results.

ANOMALIES OF FORM AND POSITION.

Congenital Anomalies of Form.—The liver, in consequence of defects of development, is occasionally found in the thoracic cavity or projecting beneath the skin near the umbilicus (hepatomphalos); in monsters the organ may be entirely absent. Small portions of aberrant hepatic tissue are occasionally found in the suspensory ligament or the adjacent connective

¹ Consult Gordon, Cammidge, Watkins, and Robson, *Lancet*, 1905, ii, 1687, 1803, 1951; 1906, i, 57, 185, 255.

² Schmidt und Strasburger, *Die Faeces des Menschen*, 1905, 2te Aufl. 225.

tissue. In partial and complete transposition of the viscera the liver may be as much in the left half of the body as normally it is in the right; in this event the left lobe is the larger. Rarely the normal relative size of the different lobes is changed: the left lobe may be unusually large or unusually small (defective blood supply during intra-uterine or early extra-uterine life); the smaller lobes may be smaller or larger than normally, and they are sometimes more or less pedunculated. Congenital linguiform lobulation of the right lobe has been described; it is commoner in children; and it may be mistaken clinically for acquired linguiform lobulation. Well-marked lobulation of the entire organ is occasionally encountered: sometimes, doubtless, it is a foetal manifestation, homologous with similar lobulation of the kidney; often, however, it is a postnatal acquired condition, due to syphilis, perhaps tuberculosis, perihepatitis, etc.; it is scarcely likely to be mistaken for the distorted pseudolobulation sometimes seen in cirrhosis.

Acquired Anomalies of Form (Deformities).—The important acquired anomalies of form consist of: (1) The so-called corset, or constricted, liver; (2) linguiform lobulation; and (3) changes due to diseases of adjacent organs.

The corset, or constricted, liver is the consequence of tight lacing, and is therefore almost wholly confined to the female sex (as much as 25 per cent. of female cadavers in some series); but an analogous condition is sometimes encountered in men, the result of the constricting influence of a tight belt or strap. The basis of the deformity is a pressure atrophy of the liver parenchyma followed by fibrosis. Depending upon the plane of maximum compression, that is the waist, which varies somewhat with changing fashions, the organ as a whole may be flattened from above downward; or it may be more globular or pyramidal in shape, that is, pushed high up in the abdomen, displacing the diaphragm, and much more of its substance being above than below the plane of constriction; in either event the right lobe, bearing the brunt of the constriction, shows the most marked changes. Sometimes there is only a deep circumferential furrow; usually the liver is considerably elongated and markedly thinned at the point of greatest constriction (most marked atrophy), which often corresponds with the plane of the upper pole of the right kidney. In some cases but little hepatic parenchyma remains at the point of maximum constriction, and the elongated portion of the right lobe, which may reach to the transverse umbilical line, is attached to the major portion of the organ by a fibrous hinge-like band. Various grades of this condition are encountered, and it gradually merges into that spoken of as a linguiform (or Riedel's) lobe (partial hepatoptosis). Occasionally the left lobe is similarly constricted and elongated. The surface of the superior part of the right lobe usually exhibits furrows or indentations corresponding with the ribs; occasionally the posterior margin of the left lobe is much infolded (corresponding folds of the diaphragm are usually encountered). The capsule of the liver and the overlying peritoneum are often much thickened, especially at the point of greatest pressure and constriction.

Linguiform lobulation is in many respects an exaggeration of the condition previously described. Both are common in female subjects, but the linguiform lobe, as originally pointed out by Riedel (whence Riedel's lobe) is intimately related to cholelithiasis and cholecystitis, enlargement of the gall-bladder being found in 60 per cent. of the cases. Tight lacing, however,

is often at least partly responsible for the condition, since it doubtless leads to kinking of the cystic duct and consequent obstruction to the free flow of bile. These linguiform lobes are often long and much attenuated; frequently they are attached to the main portion of the liver by a thin fibrous band; and they are often extremely mobile (partial hepatoptosis). In consequence of long-continued congestion they not infrequently show the lesions of well-marked nutmeg liver with increase in fibrous tissue. They are comparatively commonly the seat of other diseases, such as gumma, abscess, tumor, etc.

Diseases of the adjacent organs, notably tumors, tuberculosis, rickets, and other diseases of the vertebrae and ribs, by compression and traction (in the event of adhesions) may alter more or less and variously the shape and somewhat possibly the size of the liver. Diseases of the stomach, omentum, etc., may also act in this manner, but much less actively.

Symptoms.—The corset liver and a linguiform lobe may be entirely symptomless. In some cases there are symptoms of indigestion, as likely due to the tight lacing per se as to the changes in the liver. A lingual lobe is sometimes painful and tender, due to congestion or associated cholecystitis and cholelithiasis; occasionally nausea and vomiting ensue, and have been attributed to interference with the functions of the pylorus and duodenum; occasionally pains of biliary colic occur. In other cases the abnormality of the right lobe is detected accidentally, and may be mistaken for an abdominal tumor.

Diagnosis.—The diagnosis is not especially difficult when the likelihood of the conditions is borne in mind and the abdomen is examined carefully. Usually the abnormal lobe will be found to move with the liver during respiration, and it can be proved to be continuous with the liver, even when a coil of intestine courses over the most constricted part. The lobe may be mistaken for a dilated gall-bladder, floating kidney, hydronephrosis, tumor of the stomach, colon, pancreas, omentum, or ovary (solid or cystic), appendicitis, etc.

Prognosis.—The prognosis depends upon the etiological factors and the advance made by the lesions.

Treatment.—The corset liver is better prevented than cured, and best prevented by efforts to approximate the classic waist of the Venus de Milo. The symptoms attributable to a linguiform lobe are most amenable to the surgical measures appropriate to the cure of cholecystitis and cholelithiasis, to which they are largely due. Suturing the lobe to the abdominal wall and resection of the lobe have been practised with good result.

Acquired Anomalies of Position.—The liver may be displaced upward, downward, or laterally, or it may be rotated on its anteroposterior axis (anteverted or retroverted). Displacement downward results in consequence of disease above the diaphragm, such as pleuritis with exudation, hydrothorax, emphysema, tumors or abscesses of the lungs or mediastinum, etc., as well as of disease between the diaphragm and the liver, subdiaphragmatic or suprahepatic abscess. Displacement upward results in consequence of disease of the abdominal organs, especially ascites, tympanites, tumors, traumatic diaphragmatic hernia, etc. Frequently when displaced upward the liver is also rotated posteriorly, so that the inferior surface presents anteriorly and upward, due to the liver being rather fixed posteriorly with the inferior vena cava. Occasionally the liver is tilted anteriorly, so that much of the upper surface of the organ comes in contact

with the anterior abdominal wall; its anterior edge may fall as low as or lower than the umbilicus, whence marked enlargement of the organ may be simulated. This may occur in large aneurism of the descending thoracic or upper abdominal aorta, and in tumors of the kidney, adrenal, retro-peritoneal glands, etc. The *symptoms* of such displacement of the liver are altogether secondary to those due to the primary disorder. The *diagnosis* is usually quite obvious upon detecting the primary disorder; indeed, in some cases the evident displacement of the liver is a valuable aid in recognizing the primary disorder (such as pleural exudation, etc.). The displacement must be differentiated from other diseases causing, on the one hand, lessening of the liver dulness and apparent diminution in the size of the liver (acute yellow atrophy, portal cirrhosis, intestinal distention, etc.), and those, on the other hand, causing enlargement of the liver (fatty and amyloid degeneration, portal and biliary cirrhosis, leukæmic infiltration, carcinoma, sarcoma, etc.).

Hepatoptosis.—The important acquired anomaly of position is that variously spoken of as hepatoptosis, wandering liver, movable liver, etc., a condition in which the liver is abnormally movable and presents variously in the abdominal cavity. This is discussed elsewhere (see pages 599, 612 et seq.).

DISEASES OF THE HEPATIC ARTERY.

Dilatation.—Dilatation or enlargement of the hepatic artery occurs when the portal vein is obstructed (thrombosed or compressed), in which event the artery acts vicariously for the vein; this is seen especially when small branches of the portal vein are obstructed. The dilatation involves usually the branches of the hepatic artery, but it may extend to the main trunk. This compensatory action of the artery for the vein is never efficient. A compensatory enlargement of the hepatic artery is observed also in conditions of connective-tissue overgrowth, as in the advanced stage of cirrhosis, in tumor formations, etc. The capillaries especially are involved, but the process may extend to the main trunk. This is an important factor in causing the increased portal pressure and the consequent ascites in portal cirrhosis.

Thrombosis.—Thrombosis of the hepatic artery in man is such a rarity, not more than two cases (Ledien and Lancereaux) being on record, and one of these being doubtful, that its results are unknown. Experimental occlusion of the artery or one of its main branches in the rabbit leads to more or less widespread necrosis, an occurrence that is not observed in the dog, owing to the very generous anastomosis. Doubtless similar conditions would obtain in man, since the collateral circulation is very good.

Embolism.—Embolism of the hepatic artery is comparatively uncommon. Purely mechanical emboli are probably comparatively innocuous, on account of the free arterial anastomosis, and the part of the organ cut off from blood supply by embolism of a small branch of the hepatic artery is doubtless speedily supplied by blood from a collateral vessel. Embolism of the main trunk or principal branch has resulted in anæmic infarction and necrosis (Chiari, Lancereaux, and Rolleston). Infectious emboli are not rare; they occur in sepsis, involve the smaller branches of the artery, and give rise to multiple abscesses. Clinically the general condition usually over-

shadows the local lesions in the liver. Sarcomatous emboli involving also the arterioles and capillaries are often observed in melanotic sarcoma, especially that originating in the eye.

Arteriosclerosis.—A noteworthy degree of sclerosis of the hepatic artery and its branches is uncommon, but minor grades are sometimes observed in association with general arteriosclerosis. An endarteritis is usually present in focal fibrosis, hæmochromatosis, and syphilis; in syphilis the lesions often proceed to obliteration (endarteritis obliterans), especially about gummas.

Aneurism.—Aneurism of the hepatic artery is a rare condition. Rolland¹ in 1908 was able to collect from the literature only 40 undoubted cases. The condition is about three times as common in men as in women (30 men, 9 women, Rolland). The age incidence varies from fourteen to eighty-three years, the average of 36 cases being thirty-seven years; the average in men, however, being thirty-five years, in women forty-five years (Rolland). The assigned etiological factors are trauma, cholelithiasis (injury to the wall of the artery by the calculus), embolism (especially infectious embolism), arteriosclerosis, and syphilitic endarteritis. An aneurism has formed the wall of an hepatic abscess (analogous to aneurism in tuberculous cavities in lungs).

Pathology.—The aneurism may be extrahepatic or intrahepatic. Of the 40 cases collected by Rolland, 24 were extrahepatic, 8 intrahepatic, 2 both extrahepatic and intrahepatic (two aneurisms in each), and 6 were not definitely described. In Rolland's own case there were three aneurisms, all intrahepatic. In 16 cases the main vessel was involved; in 12, the right branch; in 3, the left branch; in 3, both branches; and in 1, the cystic artery. The aneurism may be of the true or the false variety. Of the 40 cases collected by Rolland, 32 resulted in rupture into the peritoneum, the biliary passages, the portal vein, the stomach, the duodenum, with associated rupture of the liver in the intrahepatic forms in 4 cases; 6 were found unruptured; and in 2 the condition is not known. Rolland states that the average size of the aneurism is that of a hen's egg, the extremes being that of a child's head and that of a barley-corn; that in 32 of the cases there was only one aneurism; in 5 cases, two aneurisms; in 2 cases, three aneurisms; in 1 case, several aneurisms; and in 1 the number cannot be determined.

Symptoms.—Rolland states that the three most constant symptoms are pain, jaundice, and hemorrhage. Pain is rarely absent; in typical cases it is paroxysmal and resembles that of biliary colic, being referred to the right hypochondrium and epigastrium, which are usually tender. Pain is usually absent between attacks. The pain is attributed to pressure on the hepatic plexus of nerves and to stretching of Glisson's capsule. Jaundice occurred in 16 of the 40 cases; but in most of the cases there is no statement with regard to jaundice. When present it has usually been more or less permanent, and is due to pressure on the hepatic or the common bile duct. In none of the cases did the jaundice appear to be related to the attacks of pain. Hemorrhage into the alimentary tract occurred in 17 of the 40 cases. Usually the blood reached the alimentary tract by way of the biliary ducts; rarely there was direct hemorrhage into the stomach or the duodenum; and rarely the bleeding was due to pressure on the portal vein and conse-

¹ *Glasgow Medical Journal*, 1908, lxi, 342.

quent passive congestion. Melæna is the common clinical symptom; hæmatemesis is less common. The hemorrhage may be frequently repeated. The passage by the bowel of blood casts of the folds of Kerkring shows that the bleeding has occurred into the upper part of the small intestine. Fever (as high as 104°) occurred in a few cases, usually with the paroxysmal pain, and was sometimes accompanied by rigors. The liver was enlarged in cases in which the aneurism attained a considerable size; enlargement of the gall-bladder may result from pressure on the biliary ducts, or hemorrhage into the gall-bladder. Disturbances of digestion are common, and have been attributed to the effect of local pressure by the aneurism. In no case has a pulsating tumor been detected during life (Rolland). Rupture is usually attended by the ordinary evidences of internal bleeding, and is soon followed by death.

Diagnosis.—No case has been recognized during life except at exploratory laparotomy—whereby Kehr first detected the condition. Cholelithiasis and duodenal ulcer are the most likely diagnoses. Hemorrhages are much less frequent in cholelithiasis than in aneurism of the hepatic artery; in aneurism hemorrhage may be the first symptom; in cholelithiasis it is always a late symptom, usually due to fistula formation (Mester); both conditions may occur together. The association of symptoms of duodenal ulcer and of biliary colic might suggest the diagnosis.

Treatment.—Should the condition be diagnosed, the hepatic artery or one of its branches, depending on the exigencies of the case, should be ligated. This has been done successfully by Kehr, and is justifiable, since a sufficient collateral circulation is likely to develop. Aside from operative interference, treatment is purely palliative.

DISEASES OF THE HEPATIC VEINS.

Dilatation.—Dilatation of the hepatic veins and their branches is a concomitant of disease of the heart and lungs attended by failure of the right side of the heart. The dilatation extends to the central vein of the hepatic lobule, and is often associated with thickening of the vessel wall, perivascular fibrosis, and pigmentation and atrophy of the surrounding hepatic parenchyma, conditions more fully discussed under passive congestion of the liver.

Thrombosis.—Thrombosis of the hepatic veins is a rare condition, but it may be due to: (1) Intrahepatic or extrahepatic compression of the vein or its branches by a tumor, cyst, enlarged glands, etc.; (2) chronic stricture, the consequence of adhesions (perihepatitis) near the junction of the inferior vena cava; (3) neoplastic invasion of the vessel wall, which may progress to penetration of the vessel wall; (4) acute and chronic inflammatory disease of the vessel wall, syphilis, tuberculosis, etc., usually associated with interstitial hepatitis; (5) extension of a clot partially or completely occluding the inferior vena cava; and (6) occasionally it follows, sometimes precedes, thrombosis of the portal vein. The lesions are analogous to those of chronic passive congestion. Ascites and more or less enlargement of the spleen usually develop rapidly, but the condition is scarcely susceptible of clinical recognition. It may be suspected in cases of aggravated passive hyperæmia upon the sudden onset of ascites.

Embolism.—Embolism of the hepatic veins is uncommon, and can result only from reversal of the blood stream; that is, from a retrograde movement

of the blood in the inferior vena cava or the right side of the heart. The factors that may cause such retrograde movement of the blood are: (1) Vigorous contraction of the right auricle; (2) coughing, sneezing, and other violent expiratory efforts; and (3) the forces of gravity. The embolus, reaching the vicinity of the orifices of the hepatic veins (that is, the upper part of the ascending vena cava or the right auricle), may of its own weight (in the event of stagnation of the blood) drop into, or in consequence of some one of the active factors mentioned, be forced into, the hepatic veins (very close to the heart); the progress of the embolus into the liver is further facilitated by the absence of valves in the hepatic veins. Doubtless in some cases the embolus may drop from the superior vena cava through the right auricle into the inferior vena cava and the hepatic veins. The ensuing lesions in the liver vary with the nature of the embolus, being anæmic or fibrinous (non-suppurative), suppurative, or neoplastic. The non-suppurative emboli doubtless often give rise to little if any disturbance, since whatever damage results is speedily repaired by the collateral circulation; suppurative emboli give rise to more or less extensive suppurative phlebitis and abscess formation; and neoplastic emboli result in the formation of secondary new-growths. The embolic process itself is scarcely susceptible of clinical recognition; upon the advent of the condition to which it gives rise—suppurative inflammation or tumor formation—more or less equivocal signs may develop and perhaps permit of clinical recognition.

Suppurative Phlebitis.—Suppurative phlebitis (thrombophlebitis) of the hepatic veins sometimes ensues in cases of acute infectious inflammation of the liver, suppurative cholangitis, hepatic abscess, etc., the infection being more prone to involve the hepatic veins than the branches of the portal vein, because the latter are comparatively well protected by a connective-tissue capsule. In the event of suppurative phlebitis of the hepatic veins, secondary abscesses in the lungs and general septicopyemia ensue.

Chronic Obliterating Endophlebitis.—Chronic obliterating endophlebitis of the hepatic veins, a disorder characterized by slow proliferation of the lining of the hepatic veins and consequent obstruction, is a comparatively rare affection. Hess¹ in 1905 was able to collect only 23 cases in the literature, but he is inclined to believe that many cases have been overlooked, since clinically they resemble ordinary or portal cirrhosis of the liver, and unless at the necropsy the hepatic portion of the inferior vena cava is opened and the ostia of the hepatic veins examined, the clinical diagnosis of cirrhosis of the liver is likely to be declared correct.

Etiology.—According to Hess, the disease is about equally common in the two sexes, and occurs at all ages (seventeen months to forty-five years). The etiological factors are not known; some cases have been ascribed to syphilis, but with little reason; some are believed to be an expression of a congenital anomaly; Kretz believes in the influence of trauma, a tearing of the hepatic veins by coughing, and consequent scarring of the vessel wall; other cases are ascribed to perihepatitis, hepatitis, thrombosis, and primary phlebitis. Hess supports the view of a primary chronic endophlebitis.

Pathology.—The lesions consist of slow proliferation of the lining of the hepatic veins near their ostia with consequent lessening of the lumen, which progresses until complete cicatricial obstruction ensues (firm bands extending

¹ *American Journal of the Medical Sciences*, 1905, cxxx, 986.

variable distances into the liver parenchyma resulting); old thrombi in the hepatic veins; usually a small fibrotic (often hob-nailed) liver; sometimes a large congested liver; usually chronic perihepatitis and peritonitis; ascites and other evidences of portal obstruction. Microscopically the obliteration is caused by connective-tissue hyperplasia at the venous ostia; this is continuous with that of the intima of the vena cava, and is very dense, slightly cellular, and in parts contains lacunæ filled with blood; the stenosis which exists beyond the point of obliteration is due to proliferation of the intima. The liver parenchyma shows dilatation of the central veins, atrophy of the liver cells in the centre of the lobules, increased connective tissue which runs in bands through the parenchyma, numerous new-formed biliary ducts, and scattered areas of hyperplastic hepatic parenchyma.

Symptoms.—According to Hess, the onset is generally gradual. Abdominal pain or discomfort, often most marked in the hepatic region, or an increase in the size of the abdomen, is almost invariably the first symptom. In some cases gastric symptoms occur later. Ascites develops, increases rapidly, and necessitates repeated tapings at short intervals. The fluid withdrawn presents the characteristics of a transudate, but may be hemorrhagic. Distended abdominal veins are often present; oedema of the feet and anasarca occur as late manifestations. Jaundice is rarely noted, and when present is only of slight grade. The liver is invariably enlarged, firm, and smooth in the early stage, but later becomes small and slightly nodular. The spleen is large and firm. The temperature and the urine are normal. The duration of the cases studied by Hess was rarely more than six months from the onset of the symptoms.

Diagnosis.—Not a case has been correctly diagnosed during life, most of them having been mistaken for cirrhosis of the liver, tuberculous peritonitis, carcinoma of the peritoneum, etc. Hesse believes that the disorder should be suspected when signs of cirrhosis occur in a young subject in the absence of an etiological cause for cirrhosis, and are associated with pain in the hepatic region, rapid swelling of the liver, rapid development of ascites (Quincke), and the absence of a cause for portal obstruction.

Treatment.—The treatment is palliative, and should be directed to relief of the ascites, which is discussed under cirrhosis of the liver.

DISEASES OF THE PORTAL VEIN.

Dilatation.—Dilatation of the portal vein is a part of ordinary or portal cirrhosis of the liver, and is attributable to the increased portal pressure; this is due in part probably to lessening of the portal vascular area in the liver, perhaps also to the pressure exerted by cicatrizing connective tissue, but in largest measure to the transmission to the portal vein of the pressure in the hepatic artery, through the medium of dilated capillaries.

Thrombosis.—Thrombosis of the portal vein (pylethrombosis, pylephlebitis adhesiva) is more common in males than in females. It occurs at all ages, the average age in men being forty-four and two-thirds years, and in women forty-one years (Rolleston). The thrombosis may begin in the portal radicles within the liver, in the main trunk of the vein, or in the peripheral radicles anywhere within the area of collection of the vein; and subsequently by extension the process may become widespread.

Thrombosis of the intrahepatic radicles of the portal vein is the commonest form, and is due most frequently to cirrhosis. The cirrhotic process leads to gradual obstruction and ultimate isolation of certain sections of the portal distribution, and the increased pressure induces endophlebitis, in consequence of which the contained blood coagulates. The original coagula are gradually increased in size by secondary accretions, until not infrequently they become widespread, extending even to the main trunk of the portal vein. Thrombosis within the branches of the portal vein in the liver is also found in conditions that impede the local circulation, such as: failure of the general circulation by whatever cause induced; inflammatory disorders (cholangitis, cholelithiasis, liver abscess, etc.); secondary new-growths, syphilis, etc.; in these conditions, however, the coagula, as a rule, are recent and of little clinical interest.

Thrombosis of the main trunk of the portal vein usually results from slow chronic inflammatory and degenerative processes in the wall of the vein (endophlebitis, phlebosclerosis, calcification), which in turn may be set up by a variety of processes: it may be a part of the cirrhotic process; it may be associated with chronic inflammatory processes in its vicinity, that is, in the lesser omentum and peritoneum (thickening and induration, causing kinking and constriction of the vein), in the stomach and duodenum (ulcers with surrounding inflammatory induration), in the pancreas (swelling and induration compressing and distorting the vein); it may be due also to malignant and other new-growths of the stomach, intestine, pancreas, etc. The primary growth or the metastasis to regional lymph glands (hilum of the liver, retroperitoneum, mesentery, etc.), may implicate the main trunk or one of the larger branches that go to form it, and by compression, distortion, or invasion set up thrombosis. The thrombus, once formed, may extend toward or into the liver or in a retrograde direction and become very extensive.

Thrombosis of the peripheral radicles that go to form the portal vein may be induced by inflammatory or neoplastic disease in any one of the organs of the gastro-intestinal tract, the spleen, the female genitalia, etc., that lead directly or indirectly to the portal vein. The primary coagula often increase in size by proximal accretions until they reach the main trunk; or small portions may be detached and carried to the liver, where they may set up embolism and an ultimately more or less extensive secondary thrombosis.

The most frequent causes of thrombosis in any or all parts of the portal vein are cirrhosis of the liver and intra-abdominal new-growths. There is this interesting fact in connection with these thromboses: although they commonly do not undergo suppuration, bacteria not infrequently can be cultivated from them. This suggests that bacteria of attenuated virulence may be concerned in their formation, although of course one cannot exclude the possibility of secondary infection. It is not unlikely, however, that in at least some cases the bacteria set up a primary low-grade phlebitis, whereupon thrombosis follows. In some cases suppuration does follow, as in some of the inflammatory infections within the liver and in some of the thromboses beginning in the peripheral radicles of the portal vein. It is not unlikely, then, that in some cases at least the difference between adhesive pylephlebitis and suppurative pylephlebitis is one of degree rather than of kind.

Pathology.—The thrombus may be recent or old; not infrequently it is both: the one part pale, firm, fibrous, and intimately adherent to the wall

of the vein; the other reddish, soft, and readily detached. It may completely or only partially occlude the lumen of the vein; or in the same case in one place it may completely and in another partially obstruct the lumen; that is, it may be annular, parietal, channelled, etc. The portal vein is usually thickened (phlebosclerosis); it not infrequently contains calcareous plates; and it may be represented by a firm, non-channelled fibrous cord (stricture or complete stenosis). Peripherally to the thrombus the portal vein is much dilated and tortuous, and a collateral circulation, analogous to that of cirrhosis of the liver, usually becomes established. The liver presents the appearance of the primary condition—cirrhosis, malignant disease, abscess, etc.; and sometimes, in addition, dilatation of the hepatic artery (compensatory), infarction, fatty degeneration, softening, necrosis, perhaps some fibrosis, etc. When there is thrombosis of the main trunk of the portal vein, or of the splenic vein, the spleen is enlarged and not infrequently infarcted. When the main trunk or the mesenteric veins are thrombosed, the small intestine shows hemorrhages and infarction, which may proceed to gangrene; this is usually most marked in the middle of the jejunum, which has no collateral circulation.

Symptoms.—Thrombosis of the portal vein may be entirely latent; that is, occurring usually in the course of other disorders the manifestations of the primary condition usually entirely overshadow those due to the thrombosis. Sometimes, however, to the symptoms of the primary disorder others are added that may suggest the diagnosis. In about 60 per cent. of the cases (Langdon Brown) the onset is acute; that is, although the thrombosis may be a gradual process, symptoms due to complete obstruction develop suddenly. The most marked consist of ascites, enlargement of the spleen, and various disorders of the gastro-intestinal tract. Ascites is present in 65.6 per cent. of the cases (Rolleston); it may be absent in cases that speedily terminate fatally, in those in which the mesenteric veins are involved chiefly or alone, and in certain cases of long standing in which an efficient collateral circulation has been established. As a rule, the ascites develops rapidly, becomes extreme, and speedily re-accumulates after removal. The spleen is usually enlarged, often enormously engorged; but ascites and tympanites may prevent its ready recognition, and perisplenitis with adhesions may prevent enlargement. The spleen often decreases notably in size after gastro-intestinal hemorrhage. Of the gastro-intestinal symptoms, the most important are various dyspeptic symptoms, vomiting, hæmatemesis, and melæna (capillary and venous oozing, or rupture of vessels from ulceration, etc.), diarrhœa (serous or serosanguinolent, due to increased venous pressure), symptoms of intestinal obstruction (paralysis due to hemorrhagic infarction), hemorrhoids, etc. The abdomen may reveal dilated veins. The general condition is poor; the appetite, strength, and flesh fail; the urine lessens in amount (due to lowered blood pressure); anæmia, œdema, and finally cachexia supervene; and not infrequently phenomena of toxæmia suggesting uræmia develop and the patient dies.

Diagnosis.—In most cases the diagnosis is impossible, but it is suggested by sudden onset of ascites of high-grade gastro-intestinal hemorrhage, enlargement of the spleen, abdominal pain, and symptoms of intestinal obstruction. When the intrahepatic branches or the main trunk of the portal vein are involved, the disorder can scarcely be differentiated from cirrhosis, with which it is often associated; but a very acute onset suggests the thrombosis.

Unusual enlargement of the spleen and copious hæmatemesis suggest involvement of the splenic vein; diarrhœa (serous or serosanguinolent) or abdominal pain, bloody stools, and symptoms of intestinal obstruction suggest involvement of the mesenteric veins.

Prognosis.—Many subjects die soon after the development of the thrombosis; the prognosis therefore is bad. In some cases, the process being gradual, a more or less efficient collateral circulation is established, which may prolong life for some time; in this event the duration depends largely upon the primary disorder, but it is rarely more than two years.

Treatment.—The treatment is purely palliative, and resolves itself into that of serious hemorrhages and of ascites; the ascites usually requires repeated tapping. Measures to prevent thrombosis in cirrhosis and in other conditions in which it may supervene, or to limit it when it has developed (providing it can be recognized), are of theoretical interest. Rolleston suggests studies of the coagulability of the blood and the amount of calcium salts, and, should these be found increased, the administration of citric acid in 30-grain doses three times a day to prevent increase of the thrombosis. A history of syphilis suggests the use of mercury and iodides. Operative intervention, with a view to promote the formation of adhesions about the liver, or to remove the obstruction in the portal vein, is scarcely feasible.

Embolism.—Embolism of the intrahepatic branches of the portal vein is a rather common sequence of inflammatory, suppurative, ulcerative, and neoplastic processes of the organs within the area of collection of the portal vein. The most common antecedents of such embolism are appendicitis (with suppuration or ulceration and thrombosis of its veins), ulcerative processes of the intestine, notably dysentery, and tumors of the gastro-intestinal tract; but embolism occurs also in divers disorders in which thrombosis of the hemorrhoidal, mesenteric, gastric, or splenic veins develops. The emboli usually are small, and they are of significance largely on account of the conditions in the liver to which they may give rise, notably multiple liver abscesses, or secondary neoplastic deposits.

Suppurative Pylephlebitis.—Suppurative pylephlebitis (portal pyæmia) is more common in males than in females; it is especially common in early adult life, coinciding with the age incidence of appendicitis. Like adhesive pylephlebitis, it may begin in the portal radicles within the liver, in the main trunk of the vein, or in the peripheral radicles anywhere within the area of collection of the vein; and subsequently by extension the process may become more or less widespread. Usually the process begins in the peripheral radicles, being secondary to ulcerative and suppurative processes of the gastro-intestinal tract. Of these, the most common is ulcerative or suppurative appendicitis, more particularly the cases neglected surgically, in which an abscess forms and enlarges under considerable tension, or in which infectious thromboses occur in the appendicular and the mesenteric veins. Less commonly ulcerative and neoplastic processes of the stomach and intestines, such as gastric and duodenal ulcer, typhoid, tuberculous and dysenteric ulceration of the intestine, gastro-intestinal carcinoma, fissures, ulcers and strictures of the rectum and anus, suppurative or gangrenous pancreatitis, suppuration of the mesenteric glands, suppurative disease of the female genitalia, abscess of the spleen, etc., constitute the starting point of the infective thrombosis. Occasionally, also, the process originates in operative infection of the mesenteric radicles of the portal vein (operations

on the intestine). The thrombus occasionally begins in the main trunk of the vein, sometimes in consequence of operative trauma, but usually secondarily to a local focus of suppuration—perigastric, periduodenal, subhepatic, etc.; or to gallstones in the common duct, with surrounding suppuration, etc. Suppurative pylephlebitis rather rarely begins within the liver, but it may be sequential to a liver abscess, suppurating hydatid cyst, suppurative cholangitis (with or without cholelithiasis or cholecystitis), etc. In these cases the infection is much more likely to involve the hepatic rather than the portal vein, since the latter is protected by a connective-tissue envelope; but infection of the portal vein does occur, being doubtless conveyed by the lymphatics, although likely sometimes by continuity of structure. The infecting micro-organisms are staphylococci, streptococci, the colon and typhoid bacillus, *Bacillus dysenteriae*, *Amœba coli*, etc., as well as certain anaërobic bacteria.

Pathology.—The process may be more or less localized or quite widespread. Usually the peripheral radicles and some of the main branches of the vein are involved; sometimes the process involves also the main trunk and extends into the liver; in some cases the peripheral radicles and the ramifications in the liver are implicated, the main trunk being almost if not quite free; in this event the intrahepatic lesions are embolic. The vein is enlarged, swollen, softened, congested, œdematous (panphlebitis); it is filled with a more or less softened (necrotic) sanguinolent-purulent or purulent material partially or completely obstructing the lumen. When not entirely liquefied the clot is more or less friable, occasionally free in the lumen, but usually it is more or less intimately attached to the intima. Not infrequently there is obvious purulent infiltration of the wall of the vein, and a sheath of pus or purulent material about the vein (perivascular lymph spaces) and in the adjacent connective tissue. The liver is usually enlarged and contains foci of necrosis or multiple abscesses; the spleen is enlarged and softened; not infrequently there is localized or generalized purulent peritonitis (sometimes the preceding lesion); there may be a purulent pleuritis (extension through the diaphragm); and sometimes secondary abscesses develop in different regions of the body.

Symptoms.—Since suppurative pylephlebitis is a secondary disorder, the symptoms directly attributable to it are preceded by those of the disease to which it owes its origin, notably appendicitis. The onset of the pylephlebitis is usually gradual, and is manifested by the phenomena of septicopyæmia: irregular chills, fever, and sweats, a soft compressible rapid pulse, leukocytosis, and general prostration. To these may be added extension of the abdominal pain beyond the seat of original disease, enlargement and tenderness of the liver (about one-half of the cases), enlargement of the spleen (one-fourth of the cases), etc. Vomiting, hæmatemesis, and diarrhœa are sometimes observed. Symptoms referable to various complications (suppurative pleuritis, brain abscess, etc.) may supervene. Usually the patient soon passes into a typhoid state and dies.

Diagnosis.—The diagnosis rests upon an obvious primary cause, the development of a septicopyæmic state (perhaps the detection of bacteria in the blood), and evidences of involvement of the liver (enlargement, tenderness, and jaundice). In the differential diagnosis one has to consider other forms of liver abscess and of septicopyæmia, typhoid fever, etc.

Prognosis.—The disorder is doubtless fatal in the great majority of cases, especially when the main trunk of the vein is involved or when there are

multiple abscesses in the liver. One may concede the possibility of recovery from minor lesions, but this is extremely unlikely.

Treatment.—The disease is much easier prevented than cured. Appendicitis should be promptly treated surgically, and other ulcerative or suppurative conditions of the gastro-intestinal tract should be appropriately treated, so as to prevent the formation of infectious emboli. When developed the treatment of pylephlebitis is that of liver abscess.

Chronic Endophlebitis.—Chronic endophlebitis, a process allied to arteriosclerosis, apparently may be primary or secondary. The primary process is exceptionally rare, and its etiology is ill understood; doubtless some cases are due to syphilis; in splenic anæmia, hyperplastic changes initiated in the endothelium of the blood sinuses of the spleen may gradually spread to the splenic and the portal veins, and ultimately involve the portal radicles in the liver; to this is attributed the sequential cirrhosis of the liver (Banti's disease with cirrhosis of the liver). Secondary endophlebitis of the portal vein is encountered in cirrhosis of the liver, and in advanced passive congestion—mitral and tricuspid valve disease, chronic fibroid myocarditis, emphysema, etc. In these cases it is usually attributed to the long-standing increased pressure in the portal system, but in many cases toxic products doubtless also play a part. Partial or complete thrombosis may follow chronic endophlebitis; and calcification (analogous to a similar process in the arteries) not uncommonly ensues.

DISEASES OF THE LYMPHATIC VESSELS AND GLANDS.

The lymphatic vessels participate in a number of diseases that involve the liver. Commonly they are dilated in cirrhosis and in other conditions in which the circulation of the lymph is interfered with (such as hepatic ptosis with torsion, etc.). Oertel recently has shown that inflammatory changes in and about the lymphatics are common in ordinary cirrhosis. The lymphatics also are involved in acute inflammations of the liver, such as pylephlebitis, cholangitis, and in chronic inflammations, such as tuberculosis, syphilis, chronic peritonitis, etc.; and they not infrequently show involvement in abdominal neoplasms.

Enlargement of the lymphatic glands of the portal fissure is common in acute inflammations (cholangitis, pylephlebitis, phlebitis, abscess, etc.), in chronic inflammations (tuberculosis, syphilis, cirrhosis), in degenerative processes (such as amyloid disease), infiltrative processes (such as leukaemia), and tumor formations (carcinoma and sarcoma). Its chief clinical interest lies in the fact that the enlarged glands may so press upon the biliary ducts as to impede the flow of bile and thus cause jaundice, or obstruct the circulation in the portal vein and cause ascites.

RETROGRESSIVE DISORDERS OF THE LIVER.

Atrophy of the Liver.—Atrophy of the liver is a term more or less generally but inaptly applied to conditions in which the liver becomes reduced in size. Most of these conditions, however, are degenerative and not truly atrophic in character.

Localized atrophy of the liver occurs under most diverse circumstances, generally in consequence of pressure, such as tight lacing, or belts, deformities of the chest and vertebræ (kyphosis and scoliosis), tumors and other disorders of adjacent organs, etc.; it occurs also in certain diseases of the liver itself, such as in the vicinity of tumors and cysts, in consequence of the occlusion of a branch of the portal vein or hepatic artery, and in passive congestion, amyloid disease, cirrhosis, etc. Under these varying circumstances there is a more or less localized reduction in the size of the liver, which may be: (1) Truly atrophic with subsequent replacement fibrosis, as in pressure exerted by corsets or other constrictions, etc.; or (2) atrophic and degenerative (especially fatty degeneration and necrosis), as in passive congestion, amyloid disease, cirrhosis, etc.

General atrophy of the liver may follow stenosis or occlusion of the portal vein, general hepatitis with thickening of the capsule, advanced cirrhosis, advanced passive congestion, etc.; and a reduction in the size of the liver, which, however, is not a true atrophy, follows many widespread degenerative processes, such as acute yellow atrophy, and poisoning by phosphorus, antimony, arsenic, mercury, chloroform, etc. A decrease in the size of the liver, to which the term atrophy is truly applicable, occurs in conditions of general wasting of the tissues, as in senile atrophy (senile marasmus), inanition, certain cachexias, etc. The liver in these circumstances becomes reduced in size, because of a reduction in size and possibly also in number of the liver cells, without degenerative changes in the hepatic parenchyma or noteworthy changes in the interstitial tissues. In senility the liver participates in the general atrophic process that involves also other organs, notably the heart, the general musculature, the lungs, etc. Otherwise this general atrophy of the liver is due to disturbances of metabolism brought about by: (1) Deficient nourishment, as in poverty and consequent insufficient supply of food, or inability to utilize available food, as may occur in cancerous and other obstruction of the œsophagus, stomach, etc.; or (2) excessive drain and waste attendant upon malignant growths, protracted suppuration, long-continued febrile disorders, etc.

Pathology.—The liver is reduced in size, dark in color, dense, flaccid, of increased specific gravity, and dry—changes attributable to disappearance of the parenchyma and relative increase of the connective tissue. The section surface is dry, and the acini are obviously reduced in size and perhaps in number. These changes, although widespread, are not uniform throughout the organ, the margins, as a rule, being relatively more affected than the central portion of the lobes. Microscopically the liver cells are reduced in size and number, and often contain considerable pigment. The connective tissue is usually unchanged, aside from being relatively increased; in some regions it appears to have become really increased at first sight, but these areas, on closer study, will be found to consist of the collapsed framework of lobules from which the liver cells have wholly or almost wholly disappeared.

Symptoms.—The symptoms are those of the primary disorder—senility, inanition, cachexia, malignant disease, etc. Dyspeptic symptoms are sometimes in the ascendancy, even in the absence of local disease of the gastrointestinal tract, and may possibly be due, in part at least, to deficiency of bile. The stools are likely to be light in color, and the urine to contain urobilin. The liver is reduced in size—which may be difficult to determine; it is painless and not tender.

Diagnosis.—The diagnosis is to be based upon reduction in the size of the liver, and an obvious etiological factor.

Prognosis.—The prognosis is that of the primary disease.

Treatment.—The treatment varies with that of the primary disorder, which, rather than the condition of the liver, furnishes the indications.

Parenchymatous Degeneration. — Parenchymatous degeneration (cloudy swelling) is perhaps the commonest deviation from the normal exhibited by the liver. To some extent it is a physiological process, and is seen when excessive demands are made upon the liver, after unusually generous meals, excessive alcoholic indulgence, etc. Otherwise cloudy swelling results from the action of divers poisons, bacterial and non-bacterial. The process is especially common in the infectious diseases, notably typhoid fever, pneumonia, dysentery, and others of similarly severe nature. The liver is enlarged, somewhat softened, of lessened elasticity, pale, somewhat dull and opaque (as though cooked), and on microscopic examination reveals swelling, increased granulation and opacity of the liver cells, and obscuration of the nuclei. Like changes are commonly encountered in the kidneys, the heart, etc. The condition is of secondary clinical interest, being only a part of general toxæmic processes, and subsiding with the initiating toxæmia or infection. Clinically some enlargement and occasionally tenderness of the liver may be elicited.

Gaseous or Emphysematous Liver.—Gaseous or emphysematous liver is a process characterized by the formation of numerous gaseous cysts in the liver (and other organs), and due to *Bacillus ærogenes capsulatus*. The infection is rarely primary, although the organism has been cultivated from the blood before death; usually it is secondary, and occurs at or about the agonal period or after death from other causes. The organism is usually found in association with other bacteria, and although present before death, the gas formation is a purely postmortem phenomenon. The liver is most commonly involved, but the process may be widespread. The infection usually occurs by way of the portal vein (from the intestinal tract); but it may occur by way of the general circulation, or directly from the intestine to the biliary ducts and the gall-bladder. The liver is enlarged, soft, and spongy, and is pervaded by numerous usually small gaseous cysts. The condition is chiefly of pathological interest, and is not susceptible of clinical recognition.

Fatty Liver.—Fatty liver is a comprehensive term applicable to the different conditions characterized by an excess of fat in the liver. On the one hand it includes fatty infiltration, in which there is an excessive deposit of fat in otherwise unaltered or slightly altered liver cells; and on the other hand, fatty degeneration, in which in addition to the fat there are greater or less degenerative changes in the hepatic parenchyma, perhaps the direct conversion of the liver cells into fat. Formerly the two conditions were differentiated; doubtless there are extreme cases of fatty infiltration and of fatty degeneration, but many cases partake of the nature of both processes, and, contrary to former opinions, there is now considerable trustworthy evidence to suggest at least that the fat in the liver under all circumstances is derived mainly from adipose tissue elsewhere in the body; that even in phosphorus poisoning the fat in the liver is not due, wholly at least, to conversion of the liver protein. It seems impracticable, therefore, to differentiate fatty infiltration sharply from fatty degeneration; both processes are

appropriately discussed together as fatty liver, or fatty changes in the liver.

Etiology.—The causes of fatty liver are: (1) Dietetic indiscretions and associated disorders of metabolism, notably the ingestion of too much food, particularly fat and carbohydrates; to which is usually added a sedentary life and lack of muscular exercise and of mental activity—factors that, combined, tend to produce obesity, of which fatty liver (mostly infiltration) is a conspicuous feature. The fatty deposit is ascribed to incomplete oxidation of the excessive amount of food. (2) Anæmic and cachectic conditions, such as occur in the late stages of many chronic disorders, such as tuberculosis, amyloid disease, carcinoma, etc., in which in consequence of deficient supply of blood there is deficient oxidation; but the influence of toxic substances may not be ignored. (3) Passive congestion due to a variety of causes that lead to failure of the heart—in which the fatty infiltration of the periphery of the liver lobules is in marked contrast to the congested centres (nutmeg liver). (4) Poisons of most diverse nature, notably chronic alcoholism. Alcohol seems to be a definite protoplasmic poison to the liver cells, and in addition to taking oxygen for its own combustion, gives rise to deficient oxidation of other food products. The fatty changes are often associated with cirrhosis (fatty cirrhosis). Other poisons causing fatty changes are minerals, such as phosphorus, arsenic, antimony, copper, mercury, etc.; mineral acids, such as hydrochloric, nitric, sulphuric, etc.; and other chemicals, such as chloroform, iodoform, carbolic acid, phloridzin; fungi, meat poisons, etc. (5) Certain local infections of the intestinal tract, such as the gastrointestinal catarrh of children, dysentery, etc., in which toxins are carried to the liver by the portal vein. (6) Certain general infections, notably streptococcic and staphylococcic and other pyococcic infections of long duration (prolonged suppuration), tuberculosis, etc. The exact cause of the fatty liver that occurs in about one-third of the cases of tuberculosis is not well understood, although doubtless it is in some way related to deficient oxidation. It is probably not in any way related to the excessive ingestion of fat (such as cod-liver oil) assumed by some writers, since it is often found in tuberculous subjects who have never taken cod-liver or other oils. It is not unlikely related to secondary pyococcic intoxication.

Pathology.—Usually the liver is enlarged, and may weigh 3000 to 4000 grams; it is of lessened specific gravity (sometimes floating in water), and of lessened resistance; it is normal in conformation, and has a smooth surface and rounded edges, and is pale yellowish in color. The section surface is usually pale, anæmic, obviously fatty (fat droplets adhere to the knife); in cases of passive congestion, the well-known appearances of nutmeg liver (dark centre and light periphery of the lobules) are seen; and in cases of fatty cirrhosis, the association of fibrosis is obvious. Histologically the cells at the periphery of the lobules contain fat droplets that vary much in size and displace the cellular protoplasm and nuclei, which otherwise are not notably altered; after removal of the fat the cells may revert to their normal condition. Fatty degeneration may involve any part of the liver lobule; the cells reveal minute droplets of fat throughout the protoplasm, which otherwise is markedly granular, distorted, and opaque; the nuclei, at first unaltered, soon undergo retrograde change (karyolysis, karyorrhexis, and hyperchromatosis); after removal of the fat the cells are shrunken, opaque,

and evidently the seat of retrograde metamorphosis. Both processes are often combined in varying proportions.

Symptoms.—The symptoms of fatty liver are ill defined; it is important to bear in mind that such symptoms as may be present are due to the initiating disease and the degenerative change in the liver cells rather than to the mere presence of fat. In general the symptoms are those of the primary disorder, obesity, tuberculosis, alcoholism, toxæmic conditions, etc. In perhaps the majority of cases there are no symptoms referable directly to the liver; occasionally sensations of weight may be due to the mere weight of the liver, and ill-defined dyspeptic symptoms may be attributed to functional inactivity of the liver, although they are more likely to be due to associated disorders of the stomach and intestines, improper eating and drinking, etc. The liver is enlarged and commonly palpable two or three fingers' breadth below the costal margin; excessive fatty deposit in the abdominal wall, as well as the flaccidity of the liver itself, may render the examination unsatisfactory. In thin tuberculous subjects the enlarged liver, however, is usually readily palpable. In associated cirrhosis and amyloid disease, etc., the liver is usually firm and more readily palpable.

In phosphorus poisoning the early symptoms are those of gastro-intestinal irritation (epigastric distress, nausea, and vomiting). If the patient does not die soon, there is commonly a period of quiescence, that to the unwary suggests recovery and speedy cure. At the end of two or three days, however, vomiting and epigastric distress return, jaundice supervenes, the pulse becomes weak and accelerated, the heart dilated, subcutaneous, submucous, and subserous hemorrhage develop, and the patient usually dies within a week. In most cases the liver is enlarged throughout, smooth and tender; in some cases it decreases in size before death.

Diagnosis.—Minor grades of fatty liver cannot be recognized with certainty; the more marked grades must be differentiated from other causes of enlargement. The fatty cirrhotic liver occasions the symptoms of cirrhosis, notably, hæmatemesis, ascites, and enlargement of the spleen, which do not belong to the fatty liver. The amyloid liver is much firmer than the fatty, has a more or less obvious cause, and is associated with amyloid disease elsewhere. Leukæmic enlargement of the liver is readily recognized by the blood examination. The enlarged liver of passive congestion is readily determined by recognizing an obvious cause—heart or lung disease. A displaced liver should be recognized by a careful examination and detection of factors causing displacement. The liver of phosphorus poisoning should be determined by a history, general phenomena, and the subsequent development of jaundice, which does not occur in other forms of fatty liver.

Prognosis.—The prognosis depends upon the primary cause.

Treatment.—The treatment is altogether that of the primary disorder, obesity, alcoholism, tuberculosis, etc., of which, from a therapeutic point of view, the fatty liver usually constitutes an almost negligible associate. The treatment of phosphorus poisoning has been elsewhere discussed.

Amyloid Degeneration.—Amyloid degeneration is a process characterized by the conversion of the proteins of the tissues into a structureless homogeneous substance called lardaccin.

Etiology.—Amyloid degeneration is a disease of young male subjects (usually less than thirty years); but sex and age are probably only of significance in so far as the common antecedents of amyloid degeneration are most

common in young males. In the great majority of cases amyloid degeneration follows prolonged suppuration, especially that due to syphilis and tuberculosis and involving the bones and the lungs, but also other organs, such as the kidneys, etc. Less commonly it occurs in the absence of suppuration, as in the cachexia of tertiary syphilis, chronic malaria, malignant disease, and in delayed convalescence from infectious diseases. In most cases the direct cause of the disorder seems to be the absorption of the toxic products of pyogenic cocci. In most cases of prolonged suppuration these are secondary invaders, often, however, of prime importance. Most of the cases of amyloid disease occur in association with ill-drained cavities, from which toxic absorption is continuous and considerable. Experimentally amyloid degeneration has been produced by the repeated and long-continued injection of these toxins into lower animals.

Pathology.—The relative frequency of involvement of the different organs is shown by the combined statistics of Birch-Hirschfeld, Loomis, Dickinson, Goodhart, and Turner. Of 795 cases, the spleen was involved in 585, the kidneys in 539, and the liver in 387 (Rolleston). The amyloid liver is enlarged, sometimes to four or five times its normal size; its general conformation is preserved, its edges are sharp or slightly rounded, and its surface smooth; it is pale in color, firmer than normal, of increased specific gravity and lessened elasticity. The section surface is smooth, pale grayish red in color, and peculiarly translucent, glistening, or bacony in appearance (especially apparent at the edge of the cut surface). In minor grades the individual lobules are more or less obvious, but later they lose their identity. A fresh cut surface treated with iodine strikes a mahogany-brown color, which occurs in more or less ring forms in the mid-portion of the lobules, the remaining portions (centre and periphery) of the lobules being pale yellowish or yellowish brown. Microscopically the amyloid material, a homogeneous structureless material, is first found in the subendothelial layer of the capillaries of the mid-zone of the liver lobules; later it involves other capillaries and arterioles and spreads to the media; still later similar changes occur in the small venules, and ultimately the process involves the adjacent connective tissue. On account of defective nutritive supply, and doubtless also in consequence of the direct action of toxins, the parenchymatous cells show cloudy swelling and fatty degeneration, and are ultimately removed or converted into amyloid material.

The amyloid process is variously looked upon as an infiltration and as a degeneration, the distinction between which is largely a matter of academic discussion. The amyloid material itself is a combination of chondratin-sulphuric acid and a nucleoprotein (or some form of albumin). How it is formed is not definitely known, but it is likely that the circulating toxins injuriously affect the metabolism of the cells, and ultimately compromise their integrity, so that they become converted into the amyloid material.

Symptoms.—The symptoms of amyloid liver are those of amyloid disease in general—progressive emaciation, anæmia, and debility, usually in a person the subject of chronic suppuration, or a chronic syphilitic subject, etc. The liver is found to be enlarged (it may reach much below the umbilicus); it is painless, smooth, firm, and has sharp or somewhat rounded edges. The spleen is usually enlarged and firm; often there are evidences of amyloid degeneration of the kidneys (polyuria, albuminuria, waxy casts, etc.), of the intestinal tract, (diarrhoea), and of the cardiovascular system (weak heart

low blood pressure, œdema, etc.), etc. There is no jaundice or evidence of portal obstruction, except in the event of complications, such as gummas, cirrhosis, abscess of the liver, perihepatitis, etc.

Diagnosis.—The diagnosis is comparatively easy in some cases: a history of syphilis, or of long-standing suppuration, with enlargement of the liver and spleen, albuminuria, and perhaps diarrhœa, or weak heart and œdema, is quite unmistakable. Amyloid must be distinguished from other painless enlargements of the liver. The fatty liver is usually associated with obesity, alcoholism, tuberculosis, etc., and the liver is softer. The fatty cirrhotic liver is likely to be tender, there are the antecedents of cirrhosis, and its more common phenomena. Leukæmic enlargement is readily recognized by a blood examination.

Prognosis.—The prognosis is bad, although some improvement may be effected; occasionally an enlarged amyloid liver becomes considerably reduced in size.

Treatment.—The essential part is to eliminate the cause; suppurative foci must be adequately drained and the suppuration stopped as soon as possible; syphilis should be appropriately treated. Attention to the general health is of the greatest importance, and comprises good food, fresh air, rest, hygienic surroundings, bitter tonics, alteratives (syrup of iron iodide), etc. A weak heart calls for cardiac tonics; diarrhœa for regulation of the diet, astringents, opium, etc.; and albuminuria for free action of the skin and intestines, as much rest as possible to the kidneys, etc.

Pigmentation of the Liver.—This occurs under a variety of circumstances, but in itself it is of comparatively little clinical importance. Certain extraneous pigments are sometimes found, such as coal-dust (anthracosis), stone-dust (silicosis), silver (after medicinal administration), etc. The associated changes are usually those of slight or moderate fibrosis, to which perhaps the extraneous pigmentation may be etiologically related. Of intrinsic pigmentation the most important are bile pigment and other derivatives of hæmoglobin. Normally the liver cells usually show a varying number of fine yellowish-brown granules—some iron-containing (hæmosiderin), some iron-free (hæmofuscin); these are likely to be increased in atrophy of the liver cells from senility or other causes. Bile pigments (usually bilirubin, sometimes biliverdin) are found normally in the biliary passages; in cases of obstruction to the free flow of bile, these pigments are found in excess in the biliary passages and also in the liver cells (particularly necrotic cells), and in the interstitial tissue. In malaria there occurs a deposit of fine dark-brown or brownish-black pigment granules, especially in the endothelial cells of the capillaries at the periphery of the lobules and in the perivascular connective tissue; occasionally there is some associated fibrosis, that has been described as a malarial cirrhosis. A deposit of iron-containing pigment (siderosis of the liver) is found in and about various local lesions (abscesses, gummas, scars, tumors, etc.), in chronic passive congestion, cirrhosis, pernicious anæmia, leukæmia, and hæmochromatosis; and a special metabolic pigmentation is found in melanotic sarcoma. In the nutmeg liver of chronic passive congestion, iron-free and iron-containing pigment is found in and about the central veins; it becomes quite conspicuous in the advanced stages (red atrophy), in which a large section of the central part of the lobules may show such pigmentation; later the iron reaction may no longer be obtainable. In pernicious anæmia the periphery

of the liver lobules usually contains a considerable excess of iron-containing pigment, obviously derived from the hæmoglobin of the erythrocytes set free by hæmolysis; this is found also, but in less degree, in some cases of leukæmia.

Special interest attaches to the process described as hæmochromatosis, in which the liberated hæmoglobin is deposited as pigment granules, in the skin, liver, pancreas, etc. Concurrently fibrosis of the liver and pancreas occurs, a condition spoken of as pigmented cirrhosis of the liver with diabetes, or bronzed diabetes.

Calcareous Infiltration.—Calcareous infiltration (calcification) of the liver, a condition of no clinical interest, may occur as a primary or a secondary process. Secondary calcareous infiltration occurs in a variety of chronic inflammatory processes, such as in scars of gummas, chronic abscesses, hydatid cysts, in chronic cholecystitis, etc. Primary calcareous infiltration is a pathological curiosity; it has been described as occurring rarely in association with chronic nephritis, hip disease, etc., and involving the hepatic artery.

Leukæmic Infiltration of the Liver.—The leukæmic process commonly involves the liver: the lymphocytic variety always, the myelocytic variety almost always, although the disease may be quite advanced before noteworthy changes occur in the liver. The liver is enlarged, sometimes weighing 6000 grams or more, that is, three or four times its normal size; its surface is smooth, its edge somewhat rounded, and it is of lessened resistance and density. The section surface is smooth, pale, opaque, grayish, or sometimes yellowish in color. The individual lobules are usually distinguishable, and may appear to be more widely separated than normally (periportal leukocytic infiltration). Occasionally small roundish masses (lymphomatous masses), suggesting miliary tubercles, can be detected. Microscopically there is widespread leukocytic (lymphocytic or myelocytic) infiltration of the liver; this is usually most marked in the periportal spaces, where large masses separating the lobules are formed, but it involves also the lobules, being sometimes limited to the periphery, the capillaries of which may be fairly choked with leukocytes; sometimes the entire lobule is more or less uniformly infiltrated.

Symptoms.—The symptoms are those of leukæmia, the enlargement of the liver being a part of the disease (as is enlargement of the spleen, etc.). The enlarged liver is painless, smooth, and has fairly sharp edges. Ascites, which is not rarely a terminal event in leukæmia, has been variously interpreted. In many cases it is doubtless due to the blood dyscrasia and failing cardiovascular system, part of the generalized œdema or anasarca; perhaps in some cases it is due to peritonitis, sometimes tuberculous; but it has also been attributed to obstruction or thrombosis of the portal radicles.

The *diagnosis* is readily made by an examination of the blood. Other painless enlargements of the liver must be excluded. The *treatment* is that of leukæmia.

Necrosis.—Necrosis of the liver may occur as a widespread or a limited process. The widespread process, such as occurs in thrombosis of the hepatic artery or the portal vein, in acute yellow atrophy of the liver, tropical abscess, etc., is elsewhere discussed. The limited process, so-called focal necrosis (Mallory¹), occurs under a variety of circumstances and in several forms—

¹ *Journal of Medical Research*, 1901, vi, 264; *Journal of Experimental Medicine*, 1898, iii, 611.

which, however, are of pathological rather than clinical interest. Central, peripheral, and mid-zonal forms have been described;¹ the underlying factors of the varying zonal distribution of the necroses have not been definitely determined. Most of these necroses are found in acute infections, such as typhoid fever, scarlet fever, diphtheria, pneumonia, cerebrospinal fever,² etc. In some cases, doubtless, they result from the direct action on the liver cells of bacterial toxins; in other cases they follow obstruction or occlusion of the capillaries by swollen, proliferated, or desquamated endothelial cells, transported giant (usually phagocytic) cells, fibrinoid plugs, thrombi made up of agglutinated erythrocytes, etc. Experimentally they may be induced by injection into lower animals of vegetable poisons, such as abrin and ricin (Flexner³), or of hæmagglutinins of bacterial origin or those contained in certain cytolytic immune sera (Pearce⁴) which lead to the formation in the portal radicles of thrombi made up of agglutinated erythrocytes. These have a clinical interest in that they suggest the lesions as well as the mode of development of cirrhosis of the liver.

Oertel⁵ has described a multiple non-inflammatory necrosis of the liver with jaundice, and he and Symmers⁶ have carefully described the changes that occur in these necroses in the liver.

ACUTE YELLOW ATROPHY OF THE LIVER.

Acute yellow atrophy of the liver is an acute and widespread autolytic necrosis of the liver cells, characterized clinically by jaundice, reduction in the size of the liver, and toxic disturbances of cerebration, proceeding to a fatal issue. The disease is comparatively rare. The earliest record of a case is by Ballonius (died in 1616); Best,⁷ in 1903, collected 450 cases. The best recent study is by F. W. White.⁸

Etiology.—Acute yellow atrophy of the liver is especially frequent between the twentieth and the thirtieth year (50 per cent. of the cases), and in women (two-thirds of the cases); both of these facts are explainable largely by the frequent occurrence of the disease during pregnancy. Hunter's studies show that more than 80 per cent. of the cases occur between the tenth and the fortieth year; Rolleston has collected 22 cases occurring within the first ten years of life. The influence of pregnancy is noteworthy, since almost if not quite one-half of the cases in women, that is about 30 per cent. of all cases, occur about the middle or during the latter half of pregnancy; the disease is scarcely if ever seen during the first three months of pregnancy, but sometimes it occurs during the puerperium. The onset of the disorder in pregnant women has been preceded by shock, fright, mental disturbances, worry, and anxiety (related or not to the pregnancy and expected parturi-

¹ Opie, *Journal of Medical Research*, 1904, xii, 147.

² Consult McCrae, *Journal of Pathology and Bacteriology*, 1907-8, xii, 279.

³ *Johns Hopkins Hospital Reports*, 1897, vi, 259.

⁴ *Journal of Medical Research*, 1904, xii, 329; 1906, xiv, 541. Pearce and Winne, *American Journal of the Medical Sciences*, 1904, cxxviii, 669.

⁵ *Journal of Medical Research*, 1904, xii, 75; *Journal of Experimental Medicine*, 1906, viii, 103.

⁶ *Journal of Experimental Medicine*, 1907, ix, 64.

⁷ *Thesis of the University of Chicago*, 1903.

⁸ *Boston Medical and Surgical Journal*, 1908, clviii, 729 (literature).

tion), all of which are believed to exert some etiological influence; but it is extremely unlikely that these can do more than depress somewhat the general resistance of the individual. The causative influence of pregnancy is much more likely akin to that exerted by the toxæmias of pregnancy, in which we now know the liver frequently shows degenerative changes, and in the production of which it plays an important, according to some observers the important, part. The actual etiological factor, however, is not known, since the disorder does not occur more commonly than once in 28,000 pregnancies (Braun). Various known infections exert some etiological influence; thus 10 per cent. of the cases occur during secondary syphilis (in which slight jaundice is not very rare). It is not likely, as has been contended, that the lesions in the liver are provoked by the mercury used to treat the syphilis, since syphilis and the prolonged use of mercury are very common, and acute yellow atrophy is very rare, but the fact that mercury may increase autolysis¹ may not be without significance in this connection. Veszpremi and Kanitz² believe that the lesions in the liver are due solely to the toxins of *Spirochæte pallida*, and that in a case studied by them the toxins were produced in excess by the enormous numbers of what they believe to be very virulent spirochætes in the skin of the patient. Various pyogenic infections (septicopyæmia, osteomyelitis, erysipelas, etc.), typhoid fever, malaria, diphtheria, etc., have antedated the liver disorder. Micro-organisms, especially the colon bacillus and pyococci, have been found in some cases, but being absent in most cases, it is difficult to believe that they possess etiological significance; they are probably only secondary invaders. Bacterial toxins of unknown nature and unknown source have also been postulated, but definite proof is not forthcoming. Etiological significance is also attributed to certain non-bacterial poisons, such as alcohol, ptomains, chloroform, phosphorus, etc. Chloroform is of undoubted significance, being responsible for the cases of so-called delayed chloroform poisoning.³ Phosphorus gives rise to a somewhat analogous condition, but the lesions in the liver are not alike in the two disorders. Similar phenomena may develop in the course of other diseases of the liver, such as advanced passive congestion, cirrhosis, cholangitis, etc. All of the foregoing leads inevitably to the conclusion that the designation acute yellow atrophy, as well as icterus gravis, comprises a series of diverse disorders that exhibit more or less superficial resemblance. We are now able to separate some of the cases from the main group, such as those due to syphilis, septicopyæmia, puerperal eclampsia, phosphorus poisoning, delayed chloroform poisoning, etc., but the definite etiological factor of the major group still eludes us.

Pathology.—The liver is much reduced in size and weight, being often one-half or one-third that of the normal (800 to 500 grams or less). The reduction in size is usually uniform, although occasionally more marked in the left lobe (possible site of beginning of the process); in other cases, there is more or less irregularity, due to varying grades of the destructive process or to attempts at compensatory hyperplasia. The organ is flaccid and has so lost its normal elasticity and resistance that it may be folded or bent upon

¹ See Edsall in this work, vol. i, 126.

² *Archiv für Dermatologie und Syphilis*, 1907, lxxxviii, 35 (literature).

³ Consult Wells, *Archives of Internal Medicine*, 1908, i, 589 (literature).

itself; the capsule is wrinkled, obviously too large for the reduced organ, and if not thickened shows the liver to be greenish yellow, sometimes dirty dark grayish in color, with scattered areas more reddish or reddish brown in color; often small subserous foci of hemorrhage are also visible. On sectioning the liver it is found to be dense and resistant; the section surface is mottled—yellowish or yellowish-red areas of varying size and configuration alternating and more or less gradually merging the one into the other. The yellowish coloration is due chiefly, if not entirely, to bilirubin rather than to fatty alterations (the fat content is usually not increased, and may be diminished); the reddish areas seem to be an advanced stage of the yellow, and to be more numerous or larger the longer the patient lives. The lobular markings are usually obscured—more so in the reddish areas (in which they may be not at all discernible) than in the yellowish areas; and the entire section surface appears opaque. Examination of the fresh liver tissue (scrapings of the section surface) reveals necrotic liver cells, erythrocytes, leukocytes, leucin, tyrosin, and xanthin.

Histologically the appearances vary with the stage of the disease. There is intense and widespread (not focal) destruction of the liver cells, which usually begins at the periphery and spreads to the centre of the lobules; early the cells are somewhat swollen and roundish in outline, but they soon become pale and granular, lose their outline, shrink, and stain poorly and diffusely, and the nuclei become obscured or dissolved. Usually they exhibit a yellowish color (staining with bilirubin). Ultimately the cells disappear, apparently in consequence of autolysis, and leave behind the supporting framework of the liver, granular debris, and endothelium of the capillaries (the reddish areas on macroscopic inspection). So extreme may be the destruction of the liver cells that the microscopic section can scarcely be recognized as liver tissue. Early in some cases there is more or less fatty alteration of the liver cells, but this is not a conspicuous feature of all cases; hemorrhage into the areas of degenerated cells is not uncommon. The cells lining the small bile ducts are usually hyperplastic, and later not infrequently more or less necrotic; in consequence of desquamation they may lead to obstruction of the ducts and participate in the production of jaundice. The interlobular connective tissue usually shows more or less oedema and round-cell infiltration, which may advance into the lobules and even reach the regions about the capillaries. Should the disorder be more or less protracted, a variable amount of new connective tissue is formed.

In some especially protracted cases there are conspicuous evidences of attempts at regeneration (compensatory hyperplasia) of the liver cells.¹ Large, more or less rounded, non-pigmented cells, without fat and with multiple nuclei, arranged in solid columns (analogous to the primitive tubular liver), are found amidst the collapsed fibrillovascular framework of certain lobules, or penetrating areas of granular debris; pseudobiliary canaliculi derived from the liver cells as well as the smaller bile ducts, also are often conspicuous.

All the organs are more or less bile-stained. The spleen is soft, and is slightly or moderately, but not extremely, enlarged in about two-thirds of the cases. The kidneys are enlarged, softened, pale, opaque, and often reveal small foci of hemorrhage, cloudy swelling, and fatty changes. The

¹ Consult McCallum, *Johns Hopkins Hospital Reports*, 1903, x, 375 (literature).

heart also is swollen, softened, pale, and opaque (parenchymatous and fatty degeneration). The gastro-intestinal tract shows the lesions of catarrhal inflammation. Small hemorrhages are found beneath the skin and the various serous membranes (pleura, pericardium, peritoneum, meninges) and mucous membranes (gastro-intestinal, respiratory, genito-urinary tract), and are doubtless attributable to toxic changes in the blood and blood-vessels. Hemorrhages may occur also in the brain, and degenerative changes may be seen in the spinal cord. The blood itself is more fluid than normally, coagulates with difficulty, and readily stains the endothelium and adjacent tissues.

Studies of the chemistry of the liver by A. E. Taylor,¹ Wells,² and others show that not only is the fat content not increased, but that it may be actually diminished; this especially serves to distinguish the disorder from phosphorus poisoning, of which fatty changes constitute a conspicuous feature—30 per cent., as contrasted with 5 per cent. in acute yellow atrophy. Furthermore, in phosphorus poisoning the liver is enlarged, as a rule, whereas in acute yellow atrophy it is usually lessened in size.

Pathogenesis.—There are several conspicuous features of acute yellow atrophy that endow it with special characteristics: the rapid and marked reduction in size of a large solid organ, the speedy absorption of much of the necrotic cellular debris (in contrast to other forms of necrosis), and the appearance in the urine of leucin, tyrosin, and other amino-acids, products of destructive protein metabolism that do not occur in health. These seem unquestionably to point to the activity of some poison, virulent in nature and possessing a special affinity for the liver, upon which it acts widely and intensely. The source, nature, and exact mode of action of this poison are not definitely known.

F. W. White supports the view, originally advanced by Flexner, that the process is autolytic in nature, and that it is brought about by some poison having a specific action on the liver cells whose life it destroys without injuring the proteolytic ferments they contain, whence autodigestion ensues. The leucin and tyrosin result partly from autolysis of the liver, and partly from non-conversion into urea of leucin and tyrosin formed elsewhere. Other evidence of this autolysis is found in the albuminose (decomposing protein) and purin bodies (destruction of nuclein) sometimes encountered in the urine. The reduction in the excretion of urea and the increased excretion of ammonia are interpretable as efforts on the part of nature to neutralize increased acidosis, whence the process may be looked upon as an acid intoxication. White believes that the chief symptoms of the severe intoxication are due largely to the damage done to the functions of the liver, and that the special poisonous substances are probably: (1) Nitrogenous antecedents of urea; (2) certain products of decomposition in the bowel which are normally deprived of their toxic power in passing through the liver; and (3) decomposition products of the destroyed liver tissue; and that this view is borne out by observation of other severe diseases of the liver in which little or no jaundice occurs.

The jaundice is due to obstruction of the small biliary ducts by swollen and desquamated epithelial cells and by pressure from without exerted

¹ *Journal of Medical Research*, 1902, iii, 424.

² *Journal of Experimental Medicine*, 1907, ix, 627.

by swollen and necrotic liver cells; it is contributed to by destruction of erythrocytes and the formation of bile thrombi. The presence of bilirubin in the blood and the urine suggests that there is no suppression of the bile-forming function of the liver.

Symptoms.—The initial symptoms are those of gastro-intestinal catarrh, soon (one to four days) followed by jaundice; these may last a variable period, usually five to seven days, but sometimes several weeks. During this period the disorder is usually looked upon as ordinary catarrhal jaundice (cholangitis), and as a matter of fact there is little if anything to suggest the more serious disorder. The patient complains of malaise, perhaps fugitive neuromuscular pains, poor appetite, coated tongue, occasional vomiting, constipation, etc.; there is usually moderate jaundice and bile-pigment in the urine. Soon, however, the graver symptoms supervene. The jaundice deepens, becoming dark yellowish or bronze-like, slightly greenish (in rare, extremely rapid cases jaundice may be absent); severe nausea and vomiting set in and are often associated with hæmatemesis; diarrhœa may supervene; and grave nervous symptoms ensue—headache, mental confusion, hebetude, and restlessness, soon followed by a noisy delirium, and later coma and perhaps convulsions. Hemorrhages (disordered nutrition of the vessel walls and hæmolytic action of bile salts on the erythrocytes) occur beneath the skin and into and from divers mucous membranes (intestines, urinary tract, respiratory tract, genital tract, retina, and most commonly the stomach); abortion may occur in pregnant women. Soon the patient passes into a profound typhoid state. There may be slight fever (pyococcic infection?), but usually the temperature is below normal (until shortly before death, when there may be an agonal rise); the pulse becomes rapid, feeble, and of low tension; the respirations become accelerated and irregular; the pupils dilate (said to be important, diagnostically); the tongue becomes dry, furred, and tremulous; sordes collect on the teeth and gums; and there are fibrillary twitchings of the muscles, etc.

The liver in some cases is found slightly or moderately enlarged in the early days of the disorder; this may be due to preëxisting disease (as cirrhosis), but rarely it seems to be a part of the acute yellow atrophy itself. Soon, however, the liver dulness diminishes rapidly in extent, until it may be entirely absent or constitute a small area in the axillary region; this is due to reduction in the size of the liver and to the fact that the small flabby liver falls away from the anterior abdominal wall and is replaced by the intestine. Death may occur before the liver lessens in size. The region of the liver may be tender on pressure. Slight or moderate enlargement of the spleen may be made out in about two-thirds of the cases; in the event of unusual hemorrhage or diarrhœa the spleen usually remains small.

The fæces, as a rule, are dark and offensive. The dark color in the early stages is that of normal fæces; that of the fæces late in the disease is probably due in part at least to contained blood, which obscures the paucity of the bile, since very little if any bile probably enters the intestine in the advanced stages of most cases. The stools may be light colored, especially if there be diarrhœa.

The urine, as a rule, is lessened in amount, of increased specific gravity, of increased acidity, and high colored (bile pigment and excess of urobilin); it usually contains a small amount of albumin, and hyaline, granular, fatty, and epithelial tube casts. The highly significant changes consist in the

presence of leucin, tyrosin, and the other amino-acids (largely the result of auto-digestion of the liver); sarcolactic, diacetic, and other fatty acids (lactic, acetic, butyric, and succinic); marked reduction in the amount of urea with an associated increase in the amount of ammonia and other nitrogenous substances. Sugar is rarely found (it is much more common in phosphorus poisoning). The amount of leucin and tyrosin found in the urine, of course, is no index of the amount formed in the body (in the liver and elsewhere), since both substances are comparatively insoluble, and despite considerable elimination, considerable of both substances may be found in the liver after death.

Examination of the blood often reveals a normal or an increased number of erythrocytes (usually attributed to concentration of the blood, although Grawitz and von Jaksch found the weight of the dried blood to be not above the normal); a moderate leukocytosis (up to 16,000) and a moderate reduction of the hæmoglobin (60 to 80 per cent). Bile pigments also may be found.

Occasionally there may be ascites; Tileston,¹ recently reporting a case, has collected seven others from the literature; he attributes the ascites to changes in the circulation analogous to those accountable for the ascites of cirrhosis; it has also been attributed to a concurrent nephritis (Schöppler) and to portal obstruction the result of widespread destruction of the hepatic parenchyma (Marchand). Various cutaneous eruptions in addition to hemorrhage (erythema, herpes, etc.), swelling of the joints, etc., are also occasionally observed.

Diagnosis.—In the early stages there is scarcely anything to distinguish the cases from the more innocuous catarrhal jaundice, although jaundice and an enlarged and tender liver in a pregnant woman are of portentous significance. Careful studies of the urine, especially in pregnant women, and in cases of mild jaundice, and the detection of significant urinary changes, especially the evidences of an acid intoxication, should enable us to recognize the antecedent conditions and perhaps ward off the more serious later stages. The development of nervous symptoms in any case of jaundice should always awaken suspicion. When fully developed, jaundice, vomiting, delirium (and other cerebral symptoms), lessening in the size of the liver, and the presence of leucin and tyrosin in the urine are unmistakable. The presence of leucin and tyrosin in the urine, however, is not unequivocal, since both substances may be found in small amounts in the urine in diseases in no way related to acute yellow atrophy, such as afebrile jaundice with slight enlargement of the liver, leukæmia, erysipelas, typhoid fever, etc., and lessening in the size of the liver may be missed in those cases in which there was antecedent cirrhosis (with a small liver), or when the liver is bound to the abdominal wall by adhesions.

Phosphorus poisoning may be excluded by the absence of a history of the ingestion of phosphorus (not always trustworthy), the latter development of jaundice (which in phosphorus poisoning usually supervenes after a day or two of symptoms referable to disordered stomach), decrease in the size of the liver (which usually remains enlarged in phosphorus poisoning), the more severe nervous symptoms, and the constant occurrence and larger amounts of leucin and tyrosin. Biliary cirrhosis may be excluded by the

¹ *Boston Medical and Surgical Journal*, 1908, clviii, 510.

presence of an enlarged liver with jaundice and fever, the absence of leucin and tyrosin from the urine, and the longer and more chronic course.

Prognosis.—The disease runs a somewhat variable course. In a majority of the cases death ensues before the fourteenth day; almost one-half of the cases terminate fatally in from the fifth to the tenth day; a few cases become chronic, and may last upward of four to six to eight weeks; rarely, but apparently undoubtedly, recovery may ensue, in which event the disorder is of much protracted course. Death before the fifth day is uncommon, except in pregnant women. The prognosis is extremely unfavorable, virtually fatal, the rare recovery serving rather to accentuate the ordinary course of the disease than to hold out hope of recovery in any particular case. The prognosis is especially bad in pregnant women, also in all cases, the more violent in nature and the earlier the onset of the nervous symptoms, in the event of serious renal complications, such as oliguria, albuminuria, casts, decrease in nitrogen excretion (evidences of toxic retention or acid intoxication), and in cases with severe hemorrhages. Virtually the prognosis depends upon the severity and length of the second stage of the disease; rarely this lasts more than two or at the most three days; should the symptoms be mild or abate, hope of ultimate recovery may be entertained, but usually under these circumstances the disorder becomes chronic and the patient eventually dies. The possibility of recovery, however, cannot be denied. Formerly, in the event of recovery the diagnosis was held to have been wrong; but one can conceive that the autolytic process may stop short of complete destruction of the liver, and recently the postmortem study of protracted cases has repeatedly revealed more or less extensive repair, amounting to as much as one half or two-thirds of the liver substance and a number of cases of recovery (20 to 30, White) have now been reported. Although this eventuality cannot be denied, unhappily it is unlikely in the individual case.

Treatment.—The extraordinary mortality shows the futility of our therapeutic endeavors, nor can we hope for improvement until time and research shall have disclosed the cause. Perhaps efforts to prevent or limit the formation of gastro-intestinal toxins and to favor their elimination might lessen the mortality. Perhaps it is too much to expect that we should view every case of jaundice as likely to eventuate in acute atrophy; but jaundice in a pregnant woman is assuredly a serious disorder, and should be looked upon as a possible forerunner of acute yellow atrophy. It is wise in all cases of jaundice in pregnant women (as well as in all cases of so-called catarrhal jaundice) to study the urine for evidences of acid intoxication and destructive protein metabolism, and, should these be found, to institute the appropriate treatment. Furthermore, in view of the similarity of the hepatic lesions in acute yellow atrophy, puerperal eclampsia, and delayed chloroform poisoning, chloroform and chloral should be used much more cautiously in pregnant women, especially in treating convulsions, than has been the custom in the past.

In the early stages the patient should be confined to bed. Milk is the best food, and should be diluted with some alkaline mineral water; cereals and other carbohydrates may be given in considerable amounts, since they are likely to prevent or limit the acid intoxication. The bowels should be opened freely and repeatedly, with a view to remove all noxious matters from the intestine, as well as material from which toxic substances may be

formed: calomel and the saline cathartics seem the best. Insistence should be laid upon the drinking of large amounts of water; any good water will suffice, but an alkaline mineral water is probably preferable. The water, being absorbed with noxious materials from the intestinal tract, serves to dilute them and doubtless render them less harmful to the liver, and by occasioning diuresis serves to promote elimination. Alkaline diuretics may be added to the water or given otherwise, and should serve to promote the diuresis and counteract the acid intoxication; sodium bicarbonate also might prove efficacious in the last mentioned particular. The more stimulating diuretics, such as caffeine, theobromine (and its derivatives), sparteine, etc., probably have a more limited applicability, but may prove serviceable in some cases. Enterocolysis and hypodermoclysis may be resorted to with the same object in view. Intestinal antiseptics are indicated theoretically, but since most of them effect very little intestinal antisepsis, they are of doubtful utility; probably they do no harm, so that one may use β -naphthol, salol, salicylic acid preparations, etc.

The guiding principles in the treatment of the second stage of the disease are similar to those of the first—to prevent or limit the formation and to promote the elimination of toxins. The measures mentioned should be continued, and free catharsis and free diuresis must be maintained; otherwise the treatment is purely symptomatic. Stimulants, such as alcohol, strychnine, and digitalis are useful for the general vital depression and collapse; an ice-cap to the head, the bromides internally, and a warm bath or sponge, or a small amount of morphine ($\frac{1}{32}$ to $\frac{1}{16}$ grain) or of cocaine ($\frac{1}{8}$ grain) hypodermically, may control the restlessness and delirium; warm applications to the abdomen, and cocaine, dilute hydrocyanic acid, bismuth, and carbolic acid, or small amounts of champagne, etc., may control the vomiting.

CONGESTION OF THE LIVER.

The liver, being a large organ well endowed with bloodvessels and interposed between the portal circulation and a large area of the systemic venous circulation opening almost directly into the heart, is naturally responsive to variations in the inflow of portal blood, on the one hand, and the outflow of the general systemic venous blood, on the other hand; to these must also be added its responsiveness, in common with other organs of the body, to the supply of arterial blood. The amount of blood contained within the liver, therefore, is subject to wide variations in health, and it varies also in disease with the factors upon which it is dependent. That the liver is capable of accommodating an extraordinary amount of blood is obvious from the great size it often attains when congested, and the very marked reduction in size that may follow relief of the factors provoking the congestion (such as gastro-intestinal hemorrhage, improvement in chronic heart or lung disease, etc.). The increased amount of blood, that is, the congestion or hyperæmia of the liver, may be due to increased inflow or impeded outflow of the blood—whence we may distinguish an active and a passive congestion, but neither can be looked upon as a disease entity, since in both the amount of blood in the liver is determined by diseased conditions outside the organ. The one is largely toxic in nature and usually gastro-intestinal in origin; the other is largely mechanical in nature and usually cardiac in origin.

Active Congestion of the Liver.—Etiology.—Active congestion of the liver may result from an excess of blood from the portal vein or the hepatic artery; the portal vein is the more important factor. This active congestion develops under a variety of circumstances: (1) It occurs as a physiological process during digestion, when the amount of blood carried to the liver by the portal vein is increased, lessening and finally subsiding as digestion proceeds to its completion. This normal periodic flow and ebb of blood may be disturbed and the congestion exaggerated or unduly prolonged by excessive eating and drinking, by the use of irritating food, and by the excessive use of alcohol—all of which materially increase the functional demands upon the liver. To these factors must also be added the sedentary mode of life, the lack of exercise, and the obesity common in these subjects, which together with the usual impeded breathing compromises the freedom of the circulation in the liver. (2) Various poisons, some endogenous, some exogenous, may be carried to the liver by the portal vein and provoke acute congestion. There is little doubt that many autogenic intestinal poisons, carried to the liver by the portal vein, provoke acute congestion, and that the repeated or long-continued action of these, especially those associated with chronic alcoholism, ultimately leads to cirrhosis. Certain exogenous poisons, which being ingested may reach the liver by the portal circulation, may also set up active congestion of the liver, such as ptomaines and other irritant poisons ingested preformed with the food; a single large dose of alcohol (occasional debauch) or other similar irritant; arsenic, phosphorus, and other mineral poisons; and the toxic products of certain infections that have an important if not their chief seat in the gastro-intestinal tract, such as dysentery, gastro-enteritis, typhoid fever, yellow fever, etc. (3) Active congestion of the liver may also result from the action of the toxins of certain infections that may reach the liver by way of the general circulation, such as malaria, pyococcic infections, typhoid fever, yellow fever, influenza, etc. In some of these infections the poison may reach the liver by way of the portal and the general circulation. (4) There is a group of cases presumably of active congestion of the liver attributed by men of experience, such as Cantlie and others, to the influence of "cold." These cases are extremely rare, if they occur at all in temperate zones, but they are said to be very common in the tropics where they are spoken of as "a chill upon the liver," "tropical liver." Perhaps, however, the chill is evidence of infection already accomplished, and not itself of etiological moment. (5) Since the physiological flow and ebb of the blood into the liver is doubtless controlled by the nervous system, and since irritation of the medulla (Bernard's) may cause hyperæmia of the liver (associated with glycosuria), it has been supposed, but on thread-like grounds, that reflex neurotic (vasomotor) disturbances, such as are associated with menstruation, suppressed menstruation, the climacteric, disease of the female genitalia, etc., may lead to active hyperæmia of the liver; certain evidence of this, however, is wanting.

It is obvious that many of the aforementioned factors are the causes also of inflammation and of degenerative changes in the liver, and as a matter of fact it is difficult in many cases to separate the one condition from the other; indeed, in some cases they are combined. Degenerative changes are very common in all toxæmias, and are not uncommonly associated with congestion; and congestion itself is an early stage of inflammatory conditions. In

many cases, therefore, the resulting changes in the liver represent mere gradations of the one process, which, depending upon the severity and virulence of the operating cause, in the one case ceases at congestion and subsides, and in the other progresses to inflammation.

Pathology.—The liver presents the ordinary appearances of active congestion (which, however, after death are ill marked); it is enlarged, swollen, and contains an increased amount of blood, whence it is somewhat dark reddish or reddish brown in color, and it drips blood on section. Microscopically there is dilatation of the capillaries and small vessels of the capsule of Glisson; often there is parenchymatous and sometimes fatty degeneration, occasionally pigmentation of the liver cells. Rarely there is swelling, œdema, congestion, and proliferation of the biliary ducts.

Symptoms.—The symptoms are rarely unequivocal. Most of the cases follow or are associated with dietetic indiscretions, and the symptoms are largely those of disturbances of digestion, which in general partake of the complex popularly designated “biliousness.” In consequence of an excess of food or too much alcohol, excessive demands are made upon the liver; the wheels of catabolism become clogged and the drains of excretion more or less plugged; the blood becomes surcharged with partially detoxified protein substances and effete material, and a “bilious,” “lithæmic,” or “gouty” attack supervenes. The appetite is poor, temporarily lost, the sight or thought of food often exciting disgust; there is a bad taste in the mouth and the tongue is furred; there is epigastric distress, which sometimes amounts to actual pain, and is usually aggravated by taking food; nausea is common and vomiting may supervene; there is often discomfort and sometimes actual pain in the region of the liver, which rarely is referred to the right shoulder and scapula, increased by deep breathing, sudden movement, and lying on the side; usually there is constipation and the stools may be light colored, but there may be a transitory attack of diarrhoea. The patient’s general feelings are often inexpressibly distressing; there is general malaise, headache, sometimes vertigo, a feeling of nervousness with unrefreshing sleep, irritability, mental depression, etc. Occasionally there is slight jaundice; more often there is a peculiar sallowness to the face and the general integument. The liver is found enlarged and tender, but the enlargement is not marked. The urine is commonly concentrated, of increased specific gravity, and deposits an abundant sediment of urates and uric acid; occasionally there is a transitory (toxic) albuminuria. Bile pigments are present in the event of jaundice. The disorder is usually afebrile, although now and then there may be a rise in temperature to 100° F. or thereabouts (in which event the disorder should be viewed as having progressed to acute parenchymatous hepatitis).

In another class of cases, those met with in infections, such as dysentery, etc., the constitutional symptoms are the more marked, slight or moderate fever is usually present (progression to acute hepatitis), and the local gastrointestinal symptoms are more or less in abeyance, but become aggravated during digestion. The liver is enlarged and tender.

Diagnosis.—The diagnosis is usually apparent from the etiological factors, the general disturbances of the gastro-intestinal tract, the slight jaundice, and enlargement and tenderness of the liver; by no means of minor diagnostic importance is recurrence of attacks in a person of known ill-chosen habits of eating and drinking. The diagnostic difficulties consist in determining

whether or not to the congestion permanent damage to the liver tissue has been added. With recurrence of attacks, their increasing duration, and lessening intervals, this becomes the more likely; the less the enlargement of the liver and the quicker it subsides (or returns to the normal), the more is permanent damage unlikely. In the more acute cases encountered in infections aggravation of the constitutional symptoms, together with enlargement and tenderness of the liver, usually suffice for diagnosis. In these cases there are usually parenchymatous changes in addition to the congestion.

Prognosis.—The disorder rarely lasts more than three or four days; sometimes a week. It is not dangerous in itself, but acquires its importance because of its etiological factors (especially alcohol) and its frequent recurrence in the indiscrete, in those that persist in the use of alcohol, in whom it is likely to eventuate in cirrhosis, and in those continuously exposed to other etiological factors, such as malaria, dysentery, etc., in the tropics, in whom freedom from recurrences can only be obtained by removal to temperate zones (freedom from etiological causes).

Treatment.—There is reason to believe that in those who eat and drink too much and lead a sedentary life, in the gouty, those of plethoric habit, and those formerly the subjects of chronic malaria, dysentery, etc., in the tropics, prophylactic measures may serve to ward off attacks, at least recurrences, of acute congestion of the liver; and it is by no means unlikely that appropriate energetic treatment undertaken early in cases of what cannot be called other than acute congestion would often prevent the later development of cirrhosis.

Obviously those factors known to provoke an attack of acute congestion should be avoided, and one should cultivate abstemious habits. The food should be moderate in amount and easily digested; highly seasoned articles should be avoided; alcohol should be omitted altogether or taken only occasionally as whisky in small amounts, well diluted, and with the meals. The body, especially the abdomen and back of the neck, should be protected from the action of "cold," by day as well as by night. Constipation should be overcome, preferably by laxative foods, an occasional mercurial purge, and the saline cathartics. The too common sedentary life should be at least partially replaced by activity, and the cultivation of that optimistic spirit that sees joyousness in many things in life and conduces to hearty laughter.

When the attack has developed the patient should go to bed and be kept there until convalescence is complete. The guiding principle should be to reduce the functional demands upon the liver, to eliminate all factors that might provoke or add to its congestion, and to reduce any existing intestinal catarrh. These objects are best achieved by dietary regulations. The diet should be minimal in amount and absolutely unirritating; in general it should consist exclusively of milk, which should be given at stated (rather long) intervals, and well diluted with lime-water, Vichy or other alkaline water. Whey, junket, albumin water, strained broths (without seasoning), barley water, calf's foot jelly, etc., may be given later, and, indeed, early to those that seriously object to or are inconvenienced by the milk. Gradually the diet may be augmented, at first by the addition of cereals, and then meat, etc. Alcohol must be absolutely interdicted. Plain or alkaline water should be given in large amounts throughout.

The congestion of the liver and the intestinal catarrh are favorably influ-

enced by free purgation, which is best promoted by saline cathartics. They should be given in quantity sufficient to secure free watery evacuations. The use of mercurial purges and similar cholagogues in the early stage of the disorder is ill advised, since they tend to increase the congestion of an already congested organ that requires rest; but later they are of undoubted benefit in stimulating the sluggish liver. Calomel, blue mass, rhubarb, podophyllin, colocynth, jalap, etc., will be found quite efficacious, but care should be taken that the patient does not become habituated to their use.

Ammonium chloride (20 grains thrice daily) has long enjoyed a reputation in the relief of disorders of the liver; it is in extensive use by physicians in the tropics, and is believed to control the congestion and prevent inflammation of the liver, to relieve the intestinal catarrh, and to promote the elimination (by diuresis) of retained waste products (urea, uric acid, etc.). It may be given in the early stages, when it should be combined with the alkalies (alkaline waters, sodium bicarbonate, etc.), or later, when it may be combined with nitrohydrochloric acid (highly extolled in hepatic torpidity), nux vomica, or strychnine, and a bitter tonic (gentian, columba, quassia, etc.). The distressing gastric irritability of the early stage of some cases may be controlled by sodium bicarbonate, bismuth subnitrate, and carbolic acid or creosote. Intestinal antiseptics may be useful: β -naphthol, bismuth, resorcin, salol, etc.

Local measures, such as leeches, cold compresses, etc., sometimes relieve the local discomfort, and are believed by some observers to lessen the congestion of the liver. Attempts to achieve the last-mentioned object have been made by the application of leeches about the anus (a copious hemorrhage has been followed by marked reduction in the size of the liver), and by the direct aspiration of blood from the liver with a hollow needle; two or three punctures are sometimes necessary in order to find a vein of sufficient size to permit the free flow of blood. The dangers attending the procedure are sufficiently obvious, and it is difficult to conceive the circumstances in which it might be justifiable; but Cantlie, who has practised it repeatedly, believes that the chief danger (of puncturing the inferior vena cava) is obviated by the use of a needle not more than three and one-half inches long; but in addition he sees no special danger or objection to the operation in the oozing of blood from the puncture in the liver into the peritoneal cavity—the possibility of which would make many of us pause.

Those subject to recurrences of congestion of the liver are much benefited by treatment at some of the well-known spas, such as Carlsbad, Marienbad, Vichy, Ems, etc., where, in addition to regulation of the diet and free purgation, the patients are removed from business cares and worries, subjected to the psychic influences of new scenes and modes of life, and stimulated by the sight of other sufferers like themselves improving. Many of the waters of our own country, such as Saratoga, etc., are equally efficacious, but there is little if any control of the patient at the resorts, and the accessories are largely wanting.

Passive Congestion of the Liver.—Etiology.—Passive congestion of the liver is due to factors that impede the efferent circulation of the blood; these are largely mechanical in nature and cardiac in origin (whence the name cardiac liver). The disorder occurs: (1) In failure of the heart, whether acute or chronic; it is most common as well as most advanced in the late stages of mitral valve disease (especially mitral stenosis) with consecu-

tive tricuspid insufficiency; but it is found also in primary tricuspid disease, in advanced aortic valve disease (with secondary mitral and tricuspid disorder), myocardial weakening with dilatation, pericardial adhesions, etc. (2) It occurs also in the advanced stages of such diseases of the lungs as cause increased work of the right side of the heart and ultimately lead to its failure, such as emphysema, chronic bronchitis, asthma, chronic interstitial pneumonitis, chronic adhesive pleuritis, and compression of the lungs (by pleural exudates, mediastinal tumors, aneurisms, etc.). (3) It may result also from local factors that obstruct the circulation about the junction of the hepatic veins and the inferior vena cava, of which the most important are kyphosis and scoliosis, tumors, hydatid cysts, gummas, etc., of the liver, inflammatory bands and adhesions, and kinking of the hepatic veins or the inferior vena cava by a displaced heart or a large pleural or peritoneal effusion.

Pathology.—Study of passively congested livers shows that they may be divided into three classes, more or less distinct the one from the other, but representing different stages of the same process: (1) The merely congested or engorged liver, found in cases of acute or recent heart failure (common in the infections, such as pneumonia, typhoid fever, etc.), and in chronic valvular disease with well-maintained compensation in which death has occurred suddenly or after a few days' illness only; (2) the congested nutmeg liver, found in cases of long-standing heart disease with dilatation of the right heart and tricuspid insufficiency, in which compensation, although precariously maintained, has been on the whole fairly satisfactory, and in which no serious or prolonged failure has occurred until that which leads to death; and (3) the fibrotic nutmeg liver, found also in cases of long-standing heart disease with dilatation of the right heart and tricuspid insufficiency, in which one or more severe and prolonged attacks of failure of compensation have occurred, during which likely the auricle has suffered especially and, being perhaps totally incapacitated, has permitted the full force of the ventricle to be transmitted to the hepatic lobules with consequent rupture of the venules and capillaries, laceration and destruction of hepatic parenchyma, and its repair by new-grown connective tissue (scar tissue).

The congested liver in the early stages is increased in size, depending upon the amount of contained blood; it is firmer and denser, and dark purplish or bluish in color. On section the liver in the early stages drips blood, and presents a more or less uniformly congested dark bluish or purplish color, sometimes slightly mottled with lighter areas; in more advanced stages the excess of blood is less apparent, and the organ presents the characteristics spoken of as the congested nutmeg liver; the intralobular and sublobular venules, being distended and overfilled with blood, appear as dark purplish or reddish spots (transverse section) or streaks (longitudinal section), which are in marked contrast to the pale yellowish or whitish circumferential liver cells, the seat of fatty infiltration and biliary pigmentation. When of still longer duration, the liver has decreased in size and may be less than normal; it is firmer and denser, but still presents the nutmeg appearance; about the distended central veins some fine new connective tissue, often pigmented, reddish in color, may be obvious; the hepatic veins are dilated and their walls thickened and opaque; the adjacent hepatic tissue is atrophic and pigmented, and invading it fine fibrous connective-tissue trabeculae may be detected (fibrotic nutmeg liver; red atrophy of the liver). The capsule

is sometimes thickened and opaque, especially in the event of ascites, and through it the distended subcapsular veins may be apparent. In some cases there may be a more or less universal perihepatitis, commonly associated with pericarditis and pleuritis (so-called multiple serositis).

Histologically in the early stages the intralobular venules and capillaries are dilated and overfilled with blood, the dilatation extending a variable distance toward the periphery of the lobules depending upon the degree and duration of the process. The blood content of the capillaries consists not infrequently of a disproportionate number of leukocytes, which may be interpreted as evidence of stasis of the blood. More or less extensive hemorrhage into the centre of the lobules and consequent destruction of the hepatic parenchyma is not uncommon; it is found especially in cases of acute heart failure and in cases of chronic failure of compensation terminating in a sudden complete breakdown. The hepatic cells about the central veins show a variable degree of atrophy—partly a pressure atrophy and partly doubtless nutritional, the result of insufficient oxidation. In most cases, also, there is a considerable accumulation of fat in the liver cells; in some cases this is excessive and accounts for the term fatty nutmeg liver; usually, also, the cells about the central venules contain more or less pigment derived from the hæmoglobin, usually hæmatoidin, but also hæmosiderin. In consequence of atrophy of the liver cells the supporting reticulum becomes relatively increased, and it becomes absolutely increased in consequence of new-growth (replacement fibrosis). This fibrosis is usually most marked about the central vein, and it gradually radiates therefrom, following the capillaries, throughout more or less of the lobule. The endothelial cells of the capillaries are usually swollen, hyperplastic, and somewhat desquamated, which, together with the normal absence of connective tissue about the central and sublobular venules, suggests that the new fibrous tissue beginning in the centre of the lobules owes its origin to the endothelium of the capillaries; some of it, at least, is a replacement fibrosis. There is always more or less fibrosis about the hepatic veins, which, as a rule, is proportionate to the degree and duration of the congestion. About the portal veins also some fibrosis may now and then be observed, but this is always minor in grade and altogether disproportionate to the fibrosis observed elsewhere. The capsule is usually thickened, and from this new connective-tissue trabeculae usually invade the liver a variable distance to join the new perilobular connective tissue.

The new-growth of fibrous tissue has led to its comparison with cirrhosis, and the term cardiac cirrhosis has been employed. But the lesions are in no way comparable to those of ordinary cirrhosis. The slight fibrosis common in the congested nutmeg liver is intimately related to the central and sublobular and hepatic veins; it results no doubt from the chronic distention of the veins; but the more marked fibrosis found in the fibrotic nutmeg liver is doubtless in large part a replacement or scarring process, to repair destroyed liver parenchyma. Such periportal fibrosis as may occasionally occur is purely subsidiary in degree and in importance, and may readily be accounted for by the facts that hepatic vein obstruction may lead to portal congestion, which in turn sets up chronic intestinal catarrh; that the poisons thus induced may be transported by the portal vein to the liver, and induce a mild grade of portal cirrhosis; that in many cases of chronic heart disease, alcoholism is quite common, and may set up an ordinary

cirrhosis; and finally, that many cases of ordinary portal cirrhosis have added passive congestion from final heart failure. The phenomena should be correctly interpreted; the liver in passive congestion rarely presents the lesions of true cirrhosis.

Much interest attaches to the proper interpretation of the aforementioned lesions. Of special interest is the idea advanced by Salaman¹ that the liver acts physiologically as a safety valve to the heart, and that the various changes in structure undergone after prolonged back pressure modify that safety-valve action and seriously alter the clinical course of the disease. Salaman states that the structure of the liver may be aptly compared to that of a sponge, and that in consequence of the elasticity of the supporting framework as well as of the parenchyma, the organ is capable of considerable expansion with subsequent recovery of shape, a recovery hastened by the pressure of the other abdominal organs. He points out also that the position of the opening of the hepatic veins into the inferior vena cava is such that the direct line of a backward flow from the auricle would be along the line of the veins. Experimentally Salaman found that at auricular pressure the liver accommodates from one-half to two-thirds of its volume, and at ventricular pressure it accommodates its own volume; and that when allowed to recoil, the elasticity of the liver carries it back to within one-third of its volume, and he believes that doubtless in the body, aided by the intra-abdominal pressure, the liver would at once regain its original volume. In recently congested livers the capacity to take up fluid was found to be about normal or slightly increased and the recoil was lessened; in the congested nutmeg liver the capacity to take up fluid was much reduced, while the recoil was increased; and in the fibrotic nutmeg liver the capacity to take up fluids was still further reduced, while the recoil was less than in the congested nutmeg liver. These facts warrant Salaman in deducing: (1) That under conditions of cardiac stress the liver will draw off a large volume of blood from the right auricle; (2) that after a certain time changes take place leading to fibrosis which very materially affect the distensibility of the liver; (3) that the fibrotic process, by increasing the force of the liver's recoil, prevents the use of the latter as a reservoir in which blood may accumulate; (4) that in complete compensation there is no engorgement of the liver and no back pressure; and (5) that whilst the liver may be looked upon as a sponge-like safety-valve to the heart, continued use of the safety-valve action leads to its own abolition.

The obvious inference is that congestion of the liver in the early stages of cardiac failure (whether due to endocardial, myocardial, pericardial, or pulmonary disorder) is a compensatory process; that this is especially active in acute failure, such as occurs in many infections and in the early stage of chronic valvular disease; that not until the liver has been distended to and beyond its capacity does œdema ensue; and that with the progress of the valvular defect and the gradual loss of the safety-valve action of the liver (due to the described lesions), œdema appears more and more easily.

Symptoms.—These consist of (1) those due to the primary cardiac or pulmonary disease; and (2) those due more directly to the disorder of the liver itself, which consist of local distress and gastro-intestinal phenomena.

¹ *Lancet*, 1907, i, 4.

The severity of the local subjective symptoms often depends more upon the rapidity of the development of the congestion than upon its grade; that is, subjective symptoms may be for some time in abeyance if the congestion develops slowly, although it may reach a high grade; whereas a less grade of congestion, developing rapidly, may occasion considerable distress. In the one class of cases, then, the manifestations of cardiac failure predominate; in another class, phenomena referable to the liver predominate (hepatic asystole).

Chief among the local symptoms are pain and distress or a sense of weight and heaviness in the right hypochondrium, aggravated by pressure, motion, deep inspiration, and the lateral posture. The congestion in the hepatic veins leads to congestion also in the portal tributaries, with consequent disturbances in digestion: poor appetite, epigastric distress, especially after eating, flatulence, nausea, constipation, etc. Jaundice is a common phenomenon; usually it is slight or moderate in grade, in which even the admixture of cyanosis and jaundice lends a very characteristic bluish-green or greenish-blue discoloration. The jaundice may be due to compression of the biliary capillaries and consequent interference with the flow of bile; perhaps more commonly to radicular cholangitis; or to the extension of the intestinal catarrh to the diverticulum of Vater and the common bile duct, in which event the obstruction is rarely complete; that is, some bile appears in the stools. In rare instances a terminal infection leads to marked increase of the jaundice (sometimes associated with mental symptoms, icterus gravis), doubtless due to toxic destruction of the hepatic cells. Œdema and ascites are common. The œdema is usually preceded for some time by enlargement of the liver, except in cases in which there is a sudden and severe cardiac collapse. One is warranted in the assumption that œdema does not supervene until the liver has exerted to its utmost its safety-valve action, and that the readiness with which œdema appears and disappears is a tolerably accurate indication of the functional activity of the liver in this respect. The ascites may be part of a general dropsy and due solely to the factors provoking the anasarca, in which event it is likely to be small in amount, proportionate to the œdema; or it may be disproportionate, large in amount, and require frequent tapplings; in this event it is associated with the fibrotic congested liver, or with chronic adhesive peritonitis (perihepatitis) or a more generalized peritonitis. An acute peritonitis may supervene, with audible and palpable friction.

Examination of the liver reveals it to be enlarged; the size varies from time to time, depending upon the condition of the heart. The organ may be palpable a full hand's breadth below the costal margin; it is uniformly smooth, has a sharp or slightly rounded edge, and is usually more or less tender. In advanced stages the liver becomes reduced in size (reduction in the amount of blood, cicatrization of connective tissue, atrophy of parenchyma), and may even recede beyond the costal arch, so as no longer to be palpable. The enlarged liver not infrequently pulsates, the pulsation being systolic in time and truly expansile is more likely to be found in early stages, before new-grown fibrous tissue interferes with expansion of the organ. This pulsation must be distinguished from a non-expansile pulsation transmitted to the liver by the contracting hypertrophied heart, by an abdominal aneurism, etc. The enlarged liver causes proportionate displacement and compression of adjacent organs; it may cause collapse of the base of the right

lung, with dulness on percussion and feeble or absent breath sounds (which should not be mistaken for pleural effusion).

The urine is concentrated, of high specific gravity and high color; it contains an excess of urobilin, and usually a small amount of albumin. The albuminuria is proportionate to the cardiac debility, and disappears with improvement in the cardiovascular condition.

The gastro-intestinal catarrh, interfering with the digestive and assimilative processes, leads to impaired nutrition and emaciation; to these are further added the consequences of auto-intoxication, provoked by the formation of toxic bodies in the intestinal tract that are not properly detoxified by the liver and enter the general circulation, and are not properly eliminated by the inefficient (congested) kidneys. To this may be attributed the headache and general nervousness and an aggravation of the dyspnoea, which, although due in part to the primary cardiac or pulmonary disease, is often, also, due in part to auto-intoxication (allied to uræmia).

Diagnosis.—This is usually evident from a knowledge of the primary disorder, an enlarged and tender liver, and other evidences of failing cardiac compensation. The enlarged and congested liver may usually be readily distinguished by the fact that its volume changes considerably from time to time, which does not obtain in the case of other enlarged livers, and that lessening in the size of the liver is commonly associated with other evidences of improved cardiac tone and an increase in the amount of urine. Congestion may be distinguished from cirrhosis of the liver by evidence of the primary cardiac or pulmonary disorder, other evidences of failing heart, a smooth liver, the absence of dilated abdominal veins, and by the response to medication directed to the condition of the heart; but one must remember that congestion is not uncommon in cirrhosis of the liver, and that the marked reduction in the size of cirrhotic livers is often due to lessening of congestion, rather than to atrophy of hepatic parenchyma. The smooth surface and varying size of the liver, responding to treatment directed to the heart, and the absence of cachexia will usually serve to exclude malignant disease; absence of the etiological factors (prolonged suppuration, etc.) will usually exclude amyloid disease; and an examination of the blood, etc., will exclude leukæmia.

Prognosis.—The prognosis depends altogether upon the primary disorder, upon the outlook and the response to treatment of the provoking cardiac disorder. To some extent, especially in acute and sudden failure of the heart, the congestion of the liver is doubtless a compensatory and more or less beneficent process. In chronic and advanced cases of heart disease this function becomes gradually abolished and the outlook correspondingly bad. Only the fibrotic nutmeg liver can be said of itself to influence materially the prognosis, which, from the very nature of the process, it renders very gloomy, presaging the fatal termination.

Treatment.—The treatment is that of the primary cardiac or pulmonary disorder causative of the hepatic congestion; virtually, whatever the primary disease, the treatment is that of failing cardiac compensation. The patient, of course, must be absolutely at rest, so as to reduce the work of the heart. The diet should be light and nutritious; fluids should be small in amount. Digitalis is the sovereign remedy; if care is taken to get a trustworthy preparation (preferably the infusion, but also the tincture), little cause for complaint will occur; but from time to time it may seem desirable to supple-

ment its action by such other drugs as *strophanthus*, caffeine, camphor, etc. The Addison pill, consisting of *digitalis*, squills, and mercury (calomel or blue mass), is valuable in many cases. Diuretics, such as theobromine, sparteine, the potassium salts, etc., may also be given with hope of benefit in many cases. Bleeding is often efficacious. Later, with improvement in the circulation, measures intended to improve the tone of the cardiac muscle are useful.

The hepatic congestion itself is also favorably influenced by cathartics, especially the saline cathartics, the natural aperient waters, etc., which should be given in quantity sufficient to cause free watery movements. The vegetable cathartics also are useful—*aloes*, *rhubarb*, *podophyllin*. These are especially serviceable in connection with a relatively dry diet, for several days, from time to time. Pain in the region of the liver may be relieved by hot compresses, an ice-bag, etc. General nervousness and insomnia are often markedly benefited by morphine hypodermically: $\frac{1}{32}$ to $\frac{1}{16}$ grain usually suffices and is quite as efficacious as, if not more so than, larger doses. Bromides, chloralamide, trional, veronal, and other hypnotics may be tried from time to time. Oedema may require punctures and the use of Southey's tubes, and ascites may require repeated tapplings. The tapping should not be too long delayed if the patient suffers much distress; on the other hand, it should not be performed too rapidly, nor too much fluid withdrawn, since the removal of the pressure sometimes permits the rapid absorption of toxic substances more or less dormant in the system and leads to the speedy development of uræmic manifestations.

PERIHEPATITIS.

Perihepatitis, an inflammation of the peritoneal investment of the liver, may be acute or chronic, and localized or diffuse.

Acute Perihepatitis.—**Etiology.**—Acute perihepatitis is almost, if not quite, without exception, a secondary disorder. Rarely it follows trauma of the right side (contusions, fractures of the ribs, etc., with or without contusion or laceration of the liver and its capsule); in the great majority of cases, however, the perihepatitis is secondary to disease of the liver or adjacent viscera. It occurs in association with acute congestion and inflammation of the liver (especially in the tropics); it is not uncommon in cases of long-standing passive congestion; it occurs in various forms of suppuration within the liver; it is associated with tubercles, gummas, and rapidly growing new-growths of the liver that reach to or project beyond the surface; it follows inflammatory and ulcerative processes in adjacent organs, such as ulcers of the stomach, duodenum, and other parts of the intestine, cholecystitis, pancreatitis, appendicitis, etc., in which circumstance the lesions ultimately often are those of one form of subphrenic abscess; it may be part of a general peritonitis; and it may follow disease of the thoracic viscera (pleuritis, pericarditis, etc.) through extension through the diaphragm. Acute perihepatitis is obviously always the result of infection; the common infecting agents are the pyogenic cocci (*staphylococci* and *streptococci*), the pneumococcus, the colon bacillus, etc.

Pathology.—The lesions may be localized or diffuse, and fibrinous, sero-fibrinous, or purulent in character. Early the peritoneum loses its lustre, and

becomes dull and opaque, the vessels become injected, and a small amount of serum or serofibrin is thrown out. The lesions may not extend beyond this: the serum becomes absorbed and the fibrinous exudation undergoes organization, with the ultimate formation of filamentous or dense adhesions. The lesions vary in situation with that of the primary cause: in traumatic cases, and those associated with various diseases of the liver itself (abscess, gummas, new-growths, etc.), and of the thoracic organs, the lesions are most likely to be on the anterosuperior surface of the liver, and the ultimate adhesions bind the liver to the diaphragm and the anterior abdominal wall; in the cases that follow disease of the adjacent abdominal organs the lesions are most likely to be at the contiguous, usually inferior, surface of the liver; in cases due to transport of infection from a distance (appendicitis for instance), the lesions are most common on the superior aspect of the organ; but most unlooked-for lesions sometimes occur. In other cases the lesions progress to suppuration.

Symptoms.—The important clinical phenomena consist of local pain and audible and palpable friction, which are quite similar to the analogous pleuritis. The pain is often acute and lancinating in character; it may be located anywhere in the region of the liver, but it is not infrequently referred to the margin of the ribs and the epigastrium (fairly widespread perihepatitis), or rather high up in the axilla (more localized peritonitis), and it frequently radiates to the supraclavicular fossa (said by Cantlie to be diagnostic); it is made worse by motion, such as deep inspiration, or the lateral posture, or by passive motion made by the physician. The movements of the right lower chest are restricted or abolished. Palpation reveals local tenderness and not infrequently rigidity of the upper abdominal muscles on the right side. Auscultation may reveal friction which, however, is often quite localized and very fleeting; rarely it may be palpated. Sometimes there is slight or moderate fever; there may be a short, hacking, dry cough sometimes set up by manipulations of the liver. Hiccough (suggesting involvement of the diaphragm) is not uncommon, and may be severe. Nausea and vomiting may occur in case the stomach, duodenum, or gall-bladder be implicated in the inflammation. Occasionally slight ascites develops—due probably to interference with drainage of the peritoneum through the diaphragm; this is quite independent of the marked ascites due to general peritonitis, etc.

Diagnosis.—The diagnosis is not infrequently obscured by the predominance of symptoms of the primary disorder; sometimes the lesions are found after death in cases in which no noteworthy symptoms were present during life. However, the diagnosis can usually be made by noting the symptoms of the primary disorder, and in addition local pain and tenderness, audible and sometimes palpable friction, and perhaps rigidity of the abdominal muscles of the right upper quadrant. The condition is probably most commonly mistaken for right-sided pleurisy—which may co-exist; the acute perihepatitis, however, can usually be distinguished by a knowledge of the primary (usually abdominal) disorder, the lower situation of the local signs, and the onset of severe pain, with radiation to the right infraclavicular region upon passively moving the liver forward and backward (Cantlie).

Prognosis.—The prognosis depends upon the primary or underlying condition; the acute perihepatitis (unless it progresses to suppuration), although distressing, is not in itself dangerous to life.

Treatment.—The treatment, of course, is that of the primary disease; but in some cases the phenomena of the acute perihepatitis so overshadow those of the primary disease as to furnish the indications for treatment. Pain often demands immediate attention and may be relieved by strapping the side (as for fractured ribs); this may obviate the necessity of giving opium, which should not be withheld should the pain continue. In some cases more or less relief attends the use of poultices, an ice-bag, dry cupping, leeches, and other forms of counterirritation, etc. The patient should be kept at rest and on a light diet until the acute manifestations have subsided, whereupon the treatment should again revert to that of the primary disorder.

Local Chronic Perihepatitis.—**Etiology.**—Local chronic perihepatitis not infrequently follows acute perihepatitis, particularly repeated attacks, and it is due, therefore, to the same causes. It is perhaps most commonly found in association with chronic passive congestion of the liver (long-continued expansion and contraction of the liver with consequent peripheral friction), pressure atrophy (corset liver, etc.), gummatous and tuberculous new-formations, tumors and hydatid cysts reaching the surface of the liver, chronic cholecystitis, gastric and duodenal ulcers, etc.

Pathology.—In many cases local chronic perihepatitis represents the process of healing of acute perihepatitis by the formation of adhesions, in which event the process is obsolete, non-progressive; the adhesions are firm, fibrous, and non-vascular; they are attached to, and virtually form a part of, the capsule of the liver; and they serve to bind the liver to adjacent structures and tissues—the diaphragm, the gall-bladder, the anterior abdominal wall, the stomach, duodenum, pancreas, intestine, omentum, etc. In some cases adhesions have not formed, the capsule of the liver revealing here and there more or less localized thickenings, much resembling the so-called milk spots of the pericardium. Rarely there appears to be some progression from the local to the general form of perihepatitis, but, as a rule, the two processes are quite distinct.

Symptoms.—The symptoms are scarcely, if ever, definite; but local pain, tenderness, and rigidity, particularly if recurrent, may suggest the disorder; confirmation may rarely be obtained by eliciting audible or palpable friction. In some cases, in which the nature of the disorder is in doubt, the diagnosis may become apparent by the onset of an acute exacerbation and the development of the symptoms mentioned in connection with acute perihepatitis. In the majority of cases, however, the diagnosis is not likely to be made—which is a matter of little or no moment, since the disorder is of comparatively little clinical importance.

Treatment.—The treatment is altogether that of the primary disorder. In those rare instances in which the chronic perihepatitis can be diagnosed, and the still rarer instances in which special treatment seems indicated, the measures mentioned in connection with acute perihepatitis are appropriate.

General Chronic Perihepatitis (Chronic Hyperplastic, Hyaline, or Deforming Perihepatitis; Sugar-iced Liver (Zuckergussleber); Multiple Serositis; Polyorrhymenitis; Pericarditic Pseudocirrhosis of the Liver (Pick).—General chronic perihepatitis is a curious disorder first described by Van Deen, later studied by Curschmann, who gave to it

the name of Zuckergussleber (sugar-iced liver), and more recently studied by Hale White, Nichols, Rolleston, etc.¹

Etiology.—General chronic perihepatitis is about equally common in the two sexes; it occurs at all ages, but is most common in the second and the fifth decades. The exciting causes are ill understood; most likely they are toxic in nature, but the nature and source of the toxin are not known. The rather common association of the condition with arteriosclerosis and chronic interstitial nephritis (19 of Hale White's 22 cases) suggests that the poison may be allied to that of uræmia, but this supposition is somewhat negatived by the rarity of chronic perihepatitis in arteriosclerosis and chronic nephritis. It is not improbable that the arteriosclerosis, nephritis, and perihepatitis may be due to the one cause, unknown as it may be. That the etiological factor is often widespread is shown by the common association of disease of several serous membranes (peritoneum, pericardium, and pleura). The process is best explained upon the assumption of a bacterial toxin that finds in the peritoneum (or other serous membrane) reduced in vitality from any cause (such, for instance, as arteriosclerosis and nephritis) a favorable soil for activity. In some cases this poison may be that of malaria, typhoid fever, or other infections; in other cases it may be syphilitic, although syphilis usually causes only a local perihepatitis; in other cases it is undoubtedly tuberculous, although the lesions are not those ordinarily provoked by the tubercle bacillus; but in many apparently non-tuberculous cases characteristic tuberculous lesions may be detected and the tubercle bacillus may be recovered from the lesions. Picchini well emphasizes the fact that the cases under discussion differ from the ordinary cases of serous membrane tuberculosis, in that in their etiology heredity appears to play no role; the disease does not appear to affect especially persons of tuberculous habitus; the inflammations of the serous membranes set in insidiously, much less acutely than do the ordinary cases of tuberculous inflammation; they may remain latent for a long time; and the lesions remain localized to the serous investment of the organs, and are unassociated with tuberculous lesions of the lungs, as is so common in the ordinary cases. In most cases, however, no definite etiological factor can be determined.

Pathology.—At first sight the cases appear to be separable into two classes, those in which the lesions are confined to the peritoneal investment of the liver, and those in which they involve also the serous membranes above the diaphragm (pericardium and pleura); but that this is a quite artificial division is obvious from the fact that the perihepatitis is often only part of a more generalized chronic peritonitis, and that the ultimate pathological lesions are identical, whether they begin below or above the diaphragm; the condition essentially is a chronic multiple serositis.

The lesions consist of the development of a thick encasement of fibrous connective tissue, whitish, glistening, and much resembling confectioner's icing, whence the name iced-liver, or sugar-iced liver (Zuckergussleber, Curschmann). This encasement may envelop the liver completely, or it may be limited to, or most marked upon, the upper surface; it may be 5 mm. or more in thickness; it is often of cartilagenous consistency; and it can usually

¹ A review of the literature, with a collection of cases and some personal observations, will be found in *Multiple Serositis*, by A. O. J. Kelly, *American Journal of the Medical Sciences*, 1903, cxxv, 116.

be detached in layers, leaving what appears to be a more or less intact peritoneum (this is quite characteristic, and serves to distinguish this from other forms of perihepatitis). This new connective tissue exhibits a marked disposition to cicatricial contraction, so that considerable distortion of the liver ensues, the narrower anterior margin in particular being frequently distorted. The gall-bladder is commonly embedded in dense adhesions and may be discovered and isolated only with great difficulty; usually it is collapsed and contains little or no bile, perhaps a small amount of mucus. The lesions not uncommonly involve the pleura (especially the right), and the pericardium, as already mentioned, but also other abdominal viscera; the spleen may be similarly encased in a thick, whitish envelope; the omentum may become thickened, indurated and rolled upon itself, forming a hard, almost unrecognizable mass; the mesentery may become thickened and much shortened, in consequence of which the intestine also becomes shortened and perhaps obstructed. In many cases the most widespread adhesions and distortion of the abdominal organs ensue, giving rise to what has been aptly described as deforming peritonitis. Of 110 cases collected by Picchini (50 observed by himself and 60 collected from the literature), the peritoneum was involved in all (although this was recognized clinically in only 13), being, therefore, the most frequent and most important lesion; pleuritis was present in 109 cases, in 73 of which it was bilateral; the pericardium was involved in 9 of 50 cases.

In 39 cases collected by the writer, with special reference to the association of chronic obliterative pericarditis and ascites, the pericardium was obliterated in all (more or less extensively calcified in 10); the right pleura was entirely obliterated in 17, and was partially obliterated or contained fluid in 19 (in 3 the condition of the right pleura was not stated); the left pleura was entirely obliterated in 15, it was partially obliterated or contained fluid in 16, it was normal in 3 (in 5 the condition of the left pleura was not stated); in 28 cases there was chronic perihepatitis, and in 31 cases, chronic perihepatitis or chronic peritonitis, or both.

The condition of the liver in these cases is of much interest. In many cases aside from distortion and compression, there are no noteworthy lesions whatever—a fact of much importance in determining the pathogenesis of the lesions. In the 39 cases already mentioned, the liver was reported as nutmeg in 14, cirrhotic in 8, normal aside from compression in 4, nutmeg and cirrhotic in 4, and in 3 there appeared to be only extension of connective tissue from the thickened capsule into the substance of the organ. In these cases (Pick's pericarditic pseudocirrhosis of the liver) the nutmeg appearance of the liver may well be attributed to a failing heart; these cases do not include the many in which the pericardium is not involved, and in which congestive changes in the liver are rare, unless there be a failure of the myocardium from other causes. In these cases, although a slight amount of fibrosis may extend from the thickened capsule a short distance into the substance of the liver, cirrhosis in an acceptable sense does not occur; but one must remember that thickening and adhesions of the capsule are not uncommon in advanced cirrhosis. The spleen in addition to being incased in a thick connective-tissue envelope and adherent to adjacent structures, especially in association with left-side pleuritis, is not infrequently enlarged, especially in the cases associated with chronic pericarditis (23 of the 39 cases already referred to). The kidneys usually exhibit the lesions of chronic

interstitial nephritis, more so perhaps in the cases in which the lesions are confined to the peritoneum than in those in which they involve also the pericardium and pleura; in these the kidneys usually show more or less congestion.

Microscopically the encasing is seen to consist of new-grown fibrous connective tissue, for the most part arranged in lamellæ, and the seat of advanced hyaline degeneration (whence Nichols speaks of the condition as *hyaloserousitis*); there are very few bloodvessels, and a small amount of round-cell infiltration—leukocytes and mast cells. Usually there is a more or less sharp line of demarcation between the new tissue and the underlying capsule of the liver.

As already stated the lesions may begin in any one serous membrane, and then remain localized thereto or gradually spread to the others. De Renzi believes that the peritoneum is usually involved first; then the right pleura, and then the pericardium. If, however, the right pleura should be involved first, the disease then extends to the peritoneum, thence to the left pleura and the pericardium. In some cases, however, this mode of progression of the disease is not preserved, as sometimes a pericarditis, usually not to be diagnosed, develops first. The intensity of the lesions about the liver finds its explanation in the normal circulation in the peritoneal cavity, whereby fluids and foreign particles, regardless of the position of the patient, are carried toward the upper surface of the liver; the lymphatics converge toward the suspensory ligament of the liver and the central tendon of the diaphragm. The attempt on the part of the peritoneum to remove certain noxious agents may result in partial or complete success. In the latter instance the peritoneum may be completely rid of the infective agent, which being carried to the mediastinal lymph vessels and glands, may infect the pericardium or the pleura, giving rise to a primary pleuritis or pericarditis; subsequently the peritoneum, by a descending infection, may become involved. In other cases the attempt on the part of the peritoneum to remove the infective agent being only partially successful, the region about the liver and the under surface of the diaphragm succumbs, and a primary perihepatitis occurs. Subsequently the infective agent may travel through the diaphragm and infect the pericardium or the pleura or both. The peculiarities of course and of distribution of the lymphatics of this region are also accountable for the much greater involvement of the upper surface of the liver, as contrasted with the lower surface, that is present in most cases. De Renzi states that the disease is characterized by the fact that it remains localized to the serous membranes; that it does not implicate the intra-abdominal and intrathoracic organs; that it pursues a remarkably slow and insidious course; that it gives rise to the exudation of large quantities of serofibrinous fluid; and that the fluid portion of the exudate tends to become absorbed, in consequence of which, fibrous adhesions with complete obliteration of the serous cavities result.

Symptoms.—The disorder often remains latent for many years, and may be entirely unsuspected during life; in other cases there is often a more or less clear history of the past occurrence of inflammation of one or more serous membranes; and in still other cases it is only examination of the patient that discloses the serous membrane disorder.

In the cases with manifest symptoms, the most common are sensations of fulness, oppression, and weight in the upper abdomen; or there may be

apparently a sudden onset with acute pain. The abdomen increases in size, and may attain very large dimensions—due evidently to ascites. With increase in the amount of ascites, the abdominal wall becomes tense and painful and the other subjective symptoms become aggravated. There may be slight and transitory jaundice. Later, œdema of the legs may develop.

A striking feature is the ascites, which is present whether the pericardial and pleural changes are absent, slight, or marked. Aside from the ascites, the clinical picture varies somewhat, depending upon the seat of origin of the disease. In some cases the disease is ushered in with acute pericarditis, pleuritis, or perihepatitis, which subsides and leaves the patient apparently well for a number of years. Usually, however, the lesions of the pericardium and of the pleura are latent, and the first sensible evidence of the disease is ascites. In some cases of primary pericarditis, slight and transient œdema of the legs may be present early, but this is not observed in many cases, inasmuch as the pericarditis is usually latent. When œdema is present early it usually soon subsides and does not recur until the ascites has been present for a long time or until shortly before death; except shortly before death it is inconspicuous when contrasted with the excessive ascites. Associated with early œdema, slight swelling of the spleen and disturbances of the gastro-intestinal tract may develop, but these usually subside with the œdema, and the disease, as a rule, is characterized by the entire absence of such symptoms, until failure of the heart supervenes, or until shortly before death. The ascites is characterized by the fact that it is excessive, that it necessitates repeated tapplings, that it recurs rapidly after tapping, and that it may remain stationary (not requiring tapping) for many years. Osler mentions a child in whom tapping was done 121 times, and Rumpf mentions a case in which tapping was done 301 times. The ascitic fluid is amber in color, has a specific gravity above 1015, as a rule, and contains upward of 3 per cent. of albumin—which suggests its inflammatory nature. After repeated tapplings, it may be impossible to draw off much fluid on account of the presence of many adhesions. The liver may or may not be palpable (depending upon the presence and the grade of congestion); it is usually not palpable in the late stages. The indurated and rolled up omentum may be palpable as a transverse mass; it should not be mistaken for the lower border of the liver. The general condition remains for a long time but little disturbed. The gastro-intestinal functions, as a rule, are well performed, and emaciation is long postponed. In some patients the urine may reveal the usual changes of chronic interstitial nephritis, or those of passive congestion. In other cases there are the obvious symptoms and signs of arteriosclerosis. Finally, in most cases the heart fails or death may be due to some intercurrent complication, such as pneumonia, acute peritonitis, etc.

Diagnosis.—Recurrent ascites in a person in otherwise good health, together with the disposition and ability to be up and about between tapplings, should suggest the diagnosis. Of especial diagnostic significance are: a history of a previous attack of pericarditis, pleuritis or perihepatitis; the early occurrence and subsequent disappearance of œdema of the legs; marked ascites with little or no œdema of the legs; an enlarged liver early (this may not occur), and a small and distorted but otherwise smooth liver late in the course of the disease; the absence or very late occurrence of marked enlargement of the spleen; a tendency to the repeated occurrence of attacks of pain, tenderness, rigidity, and possibly palpable and audible

friction in the right hypochondriac region—attributable to the attacks of perihepatitis; and the rapid recurrence of ascites after tapping. One should always endeavor to determine whether the lesions are confined to the region about the liver or are more widespread, involving the pericardium and the pleura. In these cases one must endeavor to dissociate the symptoms due to the serositis from those due to failure of the circulation induced by the pericardial part of the serositis and its consequences. In most of these cases prime importance attaches to the recognition of an adherent pericardium.

From cirrhosis of the liver the condition may be distinguished by the signs of adherent pericardium; the absence of the etiological factors of cirrhosis of the liver; the slow, insidious, protracted, and intermittent course; the long periods during which the ascites may remain stationary and the patient in good condition; the entire absence or transient presence of slight jaundice; the absence, in most cases, of portal congestion and gastro-intestinal disturbances; in some cases, the association of an enlarged, smooth, and firm liver with marked ascites; and the fact that in many cases the patient survives a large number of tapings. The syphilitic nature of the process may be suggested by the history of past infection and the other evidences of syphilis; but gummas may be present in the liver and be associated with perihepatitis and ascites without other evidences of the infection. The diagnosis sometimes can only be made by resort to the therapeutic test. The ordinary tuberculous peritonitis is suggested by less exudation, other tuberculous foci in the body, perhaps induration about the umbilicus, and the presence of fever. Malignant disease of the liver is suggested by irregular, nodular enlargement of the liver, evidence perhaps of primary tumor formation elsewhere in the body, and a more rapid course.

Prognosis.—The prognosis as to eventual recovery is bad, but the patient may live for many years. In more than 70 per cent. of the cases the duration is two years or more; in more than 50 per cent., four years or more; and it may be as long as sixteen years. The general health and strength are maintained for a long time, and the patient may go about his business between tapings. Sooner or later the periods between tapings become less, cardiac failure supervenes, or death results from some intercurrent infection.

Treatment.—The treatment is almost wholly symptomatic. Little can be done to influence the progress of the lesions. Diuretics may effect the discharge of the too abundant fluid or a similar object may be achieved by free catharsis. Potassium iodide and mercury may be tried when there is a trustworthy history of syphilis, as well as in doubtful cases, since they hold out some hope of benefit. Tapping must be performed when necessary; usually it has to be done frequently, and although occasionally the fluid remains at a standstill for long periods of time, tapping usually has to be resorted to at gradually decreasing intervals. Permanent drainage subjects the patients to the danger of infection and acute peritonitis. Operative interference, such as the Drummond-Talma operation, is theoretically contra-indicated, and practically has not been attended by any benefit in the few cases in which it has been tried.

ACUTE HEPATITIS.

The phenomena to which the term acute hepatitis are applicable may be divided into the suppurative and the non-suppurative forms, both of which conform to rather definite clinical and pathological types. The suppurative form comprises the different forms of abscess, suppurative pyelophlebitis, suppurative hepatic phlebitis, suppurative cholangitis, etc. The non-suppurative form comprises a series of most diverse disorders that exhibit all stages of progression, from active congestion to the inflammatory focal necroses of many infections (localized process) and the more widespread process manifested clinically as the different forms of icterus gravis and acute yellow atrophy of the liver.

Acute Non-suppurative Hepatitis.—Etiology.—Acute non-suppurative or parenchymatous hepatitis may result from the many factors that provoke active congestion of the liver and parenchymatous degeneration. The difference between these processes is one of degree only; in the individual case they are variously combined, the one or the other perhaps predominating, but they pass almost imperceptibly the one into the other, so that in many cases no sharp distinction can be drawn. The most common causes are the toxins of different infectious diseases, exogenous poisons, such as alcohol, phosphorus, and other metallic poisons, and certain endogenous autotoxins doubtless of gastro-intestinal origin, etc. The ensuing lesions vary with the virulence of the toxic cause, the duration or recurrence of its activity, and the resistance of the liver cells. As a matter of fact, acute hepatitis is uncommon in temperate climates, the different bacterial toxins rarely producing changes surpassing parenchymatous degeneration or cloudy swelling, unless we view as inflammatory the different forms of focal necrosis common in infections that sometimes are surrounded by inflammatory zones. Alcohol, especially if taken in fairly large amounts, undiluted, and at rather frequent intervals, induces, as a rule, only an active congestion, sometimes also more or less parenchymatous degeneration (and leads ultimately to cirrhosis); doubtless sometimes lesions that might correctly be described as inflammatory would be detected were the livers subjected at the time to microscopic study.

In the tropics, however, a disorder described by those of experience as acute parenchymatous hepatitis (tropical liver) is of frequent occurrence; it is most commonly provoked by malaria, but also by dysentery and perhaps other infections. It occurs especially in young adult Europeans, and not uncommonly soon (during the first year) after their arrival in the tropics. Undoubtedly factors other than malaria and dysentery are active in the etiology, although these may be the exciting cause acting upon a liver reduced in vitality. The influence of immoderate eating (especially of animal food), of drinking (especially alcohol), of exposure to cold and fatigue, may not be overlooked. These induce an active congestion, and on account of the increased functional demands reduce its vitality, and perhaps in this way render the liver especially vulnerable to the deleterious influence of the causes of malaria, dysentery, etc.

Pathology.—The lesions are most diverse, and no one description could accurately include the manifold variations, combinations of congestion, cloudy swelling, focal necroses, and the vascular and other phenomena of inflammation. The lesions vary from small focal necroses, small opaque

areas, scarcely obvious to the unaided eye, to the more complete or universal involvement of the organ. In slight or moderate cases the appearances of the liver are those of active congestion and cloudy swelling; in the more severe cases they vary in grade up to those of acute yellow atrophy. In general one may say that the liver is enlarged, swollen, softened, and rather pale in color; the section surface also is pale and opaque, and not infrequently mottled (depending upon the amount of congestion and active inflammatory phenomena). Microscopically, in addition to swelling, increased granulation and opacity of the liver cells, and obscuration of their nuclei, dilatation of the capillaries, arterioles, and venules, one encounters also fatty degeneration and pigmentation of the liver cells, thrombosis of certain capillaries and other small vessels, focal necroses, more or less round-cell infiltration (polynuclear leukocytes, plasma cells, proliferated young connective-tissue cells), swelling and desquamation of the endothelium of capillaries, and swelling, œdema, proliferation, and desquamation of the cells of the small biliary ducts. The round-cell infiltration occurs in the periportal connective tissue, in and about the foci of necroses, and sometimes more diffusely within the lobules (that is, it is pericellular). In malaria, considerable pigmentation occurs.¹ In some (protracted) cases, looked upon by many authors as a subacute form of acute yellow atrophy, there is more or less compensatory hyperplasia of the liver cells. In most cases, other parenchymatous organs, especially the kidneys and the heart, exhibit similar degenerative and inflammatory changes.

Symptoms.—The symptoms virtually are those of active congestion of the liver, of which acute hepatitis is only an advanced stage. Perhaps, although not necessarily, the symptoms are more aggravated in the inflammatory than in the merely congestive disorder; the liver pain and tenderness are likely to be more marked; nausea and vomiting are more common and usually more severe; the vomited matter partakes of the so-called bilious character (greenish, watery), and not infrequently contains blood—due usually to violent and distressing retching that provokes marked thirst and ultimately may induce marked prostration or collapse; the bowels are likely to be constipated, but the constipation may alternate with a foul, ill-smelling diarrhœa; and the nervous symptoms, such as headache, restlessness, irritability, mental depression, disturbed sleep, etc., are usually more marked. Jaundice is scarcely more common or more marked than in active congestion; usually it does not amount to more than a subicteric tint to the conjunctiva or a peculiar sallowness of the general integument. The liver is enlarged; sometimes, especially in early cases, notably so, when it may reach as low as the umbilicus; it may also be markedly increased upward, perhaps irregularly so; it is not likely to exhibit much enlargement in advanced cases (due doubtless to the presence of new-grown connective tissue—developing cirrhosis). The spleen is enlarged in the cases due to malaria, but usually of normal size or only slightly enlarged in other cases. The urine, as a rule, is concentrated, high-colored, of increased specific gravity, and deposits an abundant sediment of urates and uric acid; occasionally there is a transitory (toxic) albuminuria. Bile pigment is present in the event of jaundice. That, however, which distinguishes the

¹ See Craig in this work, vol. i, 411.

inflammatory from the congestive disorder is the presence of fever. It is true that slight fever, less than 100° , sometimes attends what is looked upon as congestion merely; but the gradual progression of the one disorder to the other is obvious, not only in the etiological factors and the general symptomatology, but also in the occurrence of fever. A temperature of 100° or more may be looked upon as conclusive evidence of the inflammatory nature of the disorder.

In the class of cases difficult to classify, but which exhibit various clinical and pathological relations and gradations to certain types of icterus gravis, the onset of the disorder is sometimes sudden and may be attended by a chill or chilliness and soon followed by fever; vomiting may set in, and may be succeeded by diarrhoea; slight (toxic or hæmolytic) jaundice is rather common; the liver becomes enlarged, sometimes irregularly so; and the constitutional symptoms are usually severe.

Diagnosis.—The diagnosis is usually apparent from the etiological factors and the general symptoms, as narrated. The diagnostic difficulties pertain to excluding other disorders, such as the different forms of suppurative hepatitis, and in the presence of jaundice, the different forms of toxic jaundice and acute yellow atrophy. In the early stages it may and frequently is quite impossible to differentiate between suppurative and non-suppurative hepatitis; in some cases time alone will bring the diagnosis. In some cases the one is a later stage of the other, and the suppuration may not be susceptible of recognition until the development of the general phenomena of the condition—irregular chills, fever, sweats, etc. In acute yellow atrophy the constitutional and nervous symptoms are more severe, the liver, at first enlarged, becomes small, the duration is less, and the outcome usually death. Phosphorus poisoning is suggested by the etiology, the enlarged liver, gastrointestinal hemorrhages, severe constitutional and nervous symptoms, etc.

Prognosis.—The first attack of acute non-suppurative hepatitis, especially as observed in the tropics, usually lasts about a week or ten days, and results in recovery; in some cases, however, suppuration ensues. The one attack, however, predisposes to subsequent attacks, and with each recurrence the course and termination are less favorable; the liver may remain more or less enlarged and gradually become cirrhotic, or, as in some primary attacks, the process may go on to suppuration.

Treatment.—The prophylaxis and treatment is that of active congestion of the liver. In addition, in those cases due to malaria, quinine should be administered in good doses; its use is often followed by a notable and rapid diminution in the size of the liver. In the event of recurring attacks the patient should remove from the tropics to a more temperate or cooler climate. Much benefit may be obtained by a course of treatment at some one of the well-known spas—Carlsbad, Marienbad, Vichy, etc.

Suppurative Hepatitis.—Suppurative hepatitis, or abscess of the liver, occurs under a variety of circumstances and in several forms. There is some justification for the common division into two classes—the large, single, so-called tropical abscess, and the small, multiple, non-tropical abscesses; but the processes are not altogether different, since the tropical abscess may be double instead of single, and even when single, by infecting adjacent liver tissue may give rise to the development of many secondary abscesses, and a number of small abscesses by peripheral extension may eventually coalesce and give rise to a large single abscess. It seems wise, therefore, to discuss

suppurative hepatitis as a single process, exhibiting peculiarities in accordance with its etiological factors and pathological anatomy.

Etiology.—Abscess of the liver is always the consequence of microörganism infection; the microörganisms are various, and they reach the liver by one of several pathways. The common causative microörganisms are the ordinary staphylococci and streptococci, *Amæba coli* (*Entamæba dysenteriae*), *Bacillus coli communis*, *Bacillus pyogenes fatidus*, *Bacillus typhosus*, *Bacillus dysenteriae*, *Bacillus pyocyaneus*, *Diplococcus pneumoniae*, *Proteus vulgaris*, *Actinomyces bovis*, etc. The infecting agent or agents may reach the liver directly, as when in consequence (1) of traumatism bacteria are introduced directly into the liver, or (2) when they reach the liver by extension of disease of adjacent organs; or the infection may be carried from afar by the blood stream, that is, (3) by the portal vein, (4) by the hepatic artery, (5) by the hepatic veins, (6) by the biliary ducts, or (7) by the lymphatics. The abscesses that result from traumatism or the extension of disease of adjacent organs are sometimes spoken of as primary, whereas those that result from the transport of infection from distant parts are called secondary abscesses; this distinction, however, is of little or no practical importance, and cannot always be maintained.

1. *Traumatism* occasionally gives rise to abscess of the liver, either directly or indirectly: (1) Directly, when a penetrating stab wound or gunshot wound affords entrance to pyogenic bacteria; (2) indirectly, when the traumatism, without laceration of the skin, causes contusion or rupture of the liver, which, constituting a *locus minoris resistentiae*, becomes vulnerable to pyogenic organisms which may be circulating in the general hepatic or the portal circulation. The resulting abscess is usually single and small or moderate in size.

2. *Disease of adjacent organs* rarely gives rise to abscess of the liver, but occasionally an ulcerative or suppurative (usually calculous) cholecystitis may perforate into the adjacent hepatic tissue and set up suppuration; or after the formation of adhesions, a gastric or duodenal ulcer or an ulcerating gastric carcinoma may invade the liver tissue and induce suppuration therein. In other cases suppuration in and about the liver is found in association with adjacent organs, such as the right kidney (suppurative nephritis, perinephritic abscess) or the right lung and pleura (abscess of the lung, suppurative pleuritis); in some cases, doubtless, the suppuration has involved the liver secondarily, but in other cases the liver likely has been the seat of primary disease; it is not always possible, however, to make the differentiation. The abscess is usually single and small, but sometimes it attains considerable, commonly superficial, size.

3. *Infection by way of the portal vein* is the commonest cause of abscess of the liver. In the great majority of cases dysentery, especially tropical or amœbic dysentery, is an antecedent condition, and the resulting abscess in the liver is spoken of as the single, tropical, or amœbic abscess. This has been elsewhere discussed.¹ Dysentery, although it plays the major role, is not the only factor in causing abscess of the liver by way of the portal circulation: the abscess may be due also to various other suppurative and ulcerative processes within the area of collection of the portal vein. Some of these have been referred to in connection with suppurative pylephlebitis; when the

¹ Consult Strong in this work, vol. i, 513.

suppuration within the liver occurs without suppurative pylephlebitis of the intrahepatic portion of the vein, the process is sometimes spoken of as portal pyæmia, but the several conditions are merely gradations of the one process, and divisions between them are mere artifice. This form of portal vein infection of the liver, whether or not it is accompanied by pylephlebitis, is characterized usually by the development of small multiple abscesses; occasionally, however, a single large abscess is produced, or it results later from the coalescence of several smaller abscesses.

The most common as well as the most important cause of this form of liver suppuration in temperate climates is appendicitis. In this the portal vein and its branches may or may not reveal thrombophlebitis, and the infection of the liver may result from extension of the thrombophlebitis, from metastases by way of the portal vein from the local disease in and about the appendix (the most common mode), or from transit of the infection through the retro-cæcal tissues to the liver. The severity and extent of the lesions in and about the appendix bear no constant relationship to the lesions in the liver, since, although the liver abscess is most likely to occur when the appendicular lesions are suppurative and the abscess under considerable tension, it may follow simple and more or less latent appendicitis. Other gastro-intestinal disorders also occasionally engender abscess of the liver, such as gastric and duodenal ulcers (transport of infection by the portal vein in the absence of adhesions), pyococcic, typhoid, tuberculous, actinomycotic, and neoplastic ulcers of the small and large intestine and the rectum, suppurative lesions of the pelvic organs (male and female), hemorrhoids, and infected thrombi following operations upon the intestine, rectum, anus, etc.

A solitary abscess of the liver may be a hydatid cyst, in which secondary infection has occurred.

4. *Infection by way of the hepatic artery* is a rather uncommon cause and gives rise to small multiple so-called septicopyæmic abscesses. These occur in pyococcic infections, especially infective endocarditis, purulent and fetid bronchitis, bronchiectasis, abscess and gangrene of the lungs, purulent pleuritis, suppurative periostitis, suppurative otitis media, infected operative or non-operative wounds, etc. These processes give rise to many infectious emboli which ultimately become arrested in the liver and elsewhere, where they set up multiple foci of suppuration. Sometimes the suppurative foci are found only in the liver, a fact difficult to explain, especially in those not uncommon cases in which the liver abscess follows wounds and suppuration in and about the head. One may assume that the liver in these cases is a locus minoris resistentiæ, that the bacteria may have passed through the pulmonary circulation without setting up inflammatory phenomena there, and that those bacteria which become lodged in other organs were there destroyed.

5. *Infection by way of the hepatic veins* doubtless occurs under exceptional circumstances, as in certain cases of suppuration of the head just alluded to, in which emboli may drop from the superior vena cava through the right auricle into the inferior vena cava and the hepatic veins. Perhaps it occurs also in other cases in which pyogenic bacteria from a suppurative focus in the pelvis or lower extremities, coming up the ascending vena cava, are forced into the hepatic veins by some violent motion and by a retrograde process infect the liver. This process is most likely to occur in association with myocardial weakness and venous congestion. The suppurative process

in the liver begins in and about the central veins and gradually spreads to the adjacent tissue, giving rise to multiple small abscesses, which may or may not become confluent.

6. *Infection by way of the biliary ducts* gives rise to abscess formation in the liver and in one of three ways: (1) By direct transport of the infection by way of the biliary passages themselves, such as may occur in infectious or suppurative cholangitis and pericholangitis, with extension to the adjacent interlobular tissues and the formation of multiple small abscesses; (2) by the extension of the infection in the biliary passages through the biliary mucosa to the subjacent vessels, thence to the portal vein (into which they empty), and the production of suppurative pylephlebitis, in which event the process is analogous to that described in connection with infection by way of the portal vein; and (3) by direct extension of ulcerative and suppurative processes in the biliary tract (usually calculous cholecystitis) to the adjacent liver tissue.

7. *Infection by way of the lymphatics* may cause abscess of the liver under circumstances analogous to those that occasion infection by way of the portal circulation. In some cases, doubtless, the portal vein becomes involved after the infection has first gained the regional lymphatics; in other cases in which liver abscess, especially suprahepatic abscess, follows peritoneal disorders, the infection has followed the pathway of the lymphatics which converge from the peritoneum to the upper surface of the liver; it has been suspected that in some cases of amœbic abscess of the liver the amœbæ may reach the liver by traversing the peritoneum. In some cases of abscess of the liver following suppuration of adjacent organs the infection is doubtless transported by the lymphatics.

Pathology.—The appearances of the liver in suppurative hepatitis vary with the pathway of infection, the nature and virulence of the infecting agent, and the situation and number of the abscesses. Abscesses of the liver have been variously classified; thus, from an etiological or pathological point of view, some observers speak of tropical abscesses and of pyæmic abscesses, the tropical being the so-called single or amœbic abscesses, the pyæmic, the smaller multiple abscesses. Although at first sight these two classes seem distinctive enough, no sharp line of demarcation can be drawn between them, since a tropical or amœbic abscess may be double or multiple and these later may coalesce to form a single large abscess; other multiple abscesses may also coalesce to form a large single abscess; and in the absence of definite knowledge of the etiological factor it is not always possible clinically to distinguish the one variety from the other; the differences, as a rule, are more or less accidental, and the processes in most regards represent different grades of the same process.

Cantlie has suggested a classification that has much to commend it: the basis of this is the anatomical situation of the abscess, whence he distinguishes: (1) Suprahepatic abscess, a collection of pus between the layers of the broad (coronary) ligament of the liver, bounded above by the diaphragm and below by the liver where it is destitute of peritoneal covering; this form of abscess is due to inflammation of the lymphatic vessels of the upper surface of the liver, and is independent of dysentery and other intestinal lesions; (2) intrahepatic abscess, a collection of pus within the liver substance, which comprise the great majority of cases; and (3) subhepatic abscess, a collection of pus beneath the under surface of the liver and the

peritoneum, pointing in the epigastrium, and due to a lymphangitis in the subhepatic region.

Abscesses the result of traumatism and of ulcerative and suppurative processes in adjacent organs are usually single and small or moderate in size, although those due to disease of adjacent organs sometimes attain considerable, usually superficial, extent. The appearances are similar to those of abscess formation in other parts of the body: a focus of inflammation surrounded by an area of intense hyperæmia; in the centre or in several points near the centre liquefaction necrosis of the inflammatory exudation begins and spreads by peripheral extension until a smaller or larger area of softened or fluid purulent material surrounded by a more or less well-defined limiting zone results. The softened material consists of polynuclear leukocytes, other forms of leukocytes and erythrocytes, necrotic and degenerated liver tissue, fat droplets, and detritus. The limiting zone is that common to abscesses generally, and consists of flattened, more or less degenerated and necrotic liver cells, infiltrated with leukocytes, and at the outer part, of congestion and fibroblastic proliferation. Inasmuch as these abscesses usually are situated near and involve some one of the surfaces of the liver, there is commonly a more or less extensive perihepatitis, adhesions binding the liver to the diaphragm or the adjacent organs. In some cases the collection of pus is on rather than within the liver, and merits the designation suprahepatic or infrahepatic abscess.

In the small multiple metastatic abscesses that are not uncommon in general arterial pyæmia (hepatic artery infection) or portal pyæmia (portal vein pyæmia), and sometimes occur in retrograde emboli (hepatic vein infection), or follow suppurative cholangitis, the liver is usually enlarged, swollen, and opaque, and presents the ordinary evidences of parenchymatous degeneration or cloudy swelling. In addition, the organ reveals beneath the translucent capsule, but especially on section, numerous grayish or yellowish, softened spots, surrounded by a hyperæmic zone; the spots vary much in size, some being not more than 1 to 2 mm. in diameter and not disturbing notably the consistency or lobular structure of the liver; others may be much larger, up to 5 or 10 cm. in diameter, especially in advanced stages of the process, obviously softened or diffuent, discharging their contents on section, and revealing variously sized cavities with irregular ragged necrotic walls. In some cases the smaller primary abscesses by peripheral extension have so increased in size as to form a large, many-chambered, or alveolar abscess; this may be so large as to occupy almost, if not quite, an entire lobe of the liver; or the process may involve the whole organ. The purulent contents may be thick, creamy, and yellowish, thinner and seropurulent, or stained with blood or bile (yellowish or greenish), in which event there may be also a greenish tint to the surrounding more or less necrotic liver tissue. Sometimes the pus is extremely offensive, especially if associated with ulcerative or gangrenous processes in the intestine. In some cases the larger branches of the portal vein may be obviously the seat of suppurative thrombophlebitis; in other cases pus may be found in the larger biliary ducts; in the cases due to hepatic artery or hepatic vein infection the larger branches of these vessels rarely show any noteworthy changes. Microscopically one may observe marked dilatation of the capillaries, and in some cases, in which death has occurred early, foci of coagulation necrosis, considerable leukocytic infiltration, vascular thrombosis, and perhaps bacteria—

which are to be looked upon as the earliest stage of the process that will progress to suppuration. Considerable leukocytic infiltration ensues, and it varies in situation and distribution depending upon the pathway of infection. If infection has occurred by way of the portal vein, the leukocytes accumulate around its small interlobular branches and then penetrate the intralobular branches and collect about the central veins; in arterial infection there is perhaps earlier and more marked leukocytic infiltration of the lobules and less interlobular accumulation; when infection has occurred by way of the bile passages the purulent infiltration occurs first about the finer branches of the biliary ducts; and in those cases in which there has been a retrograde infection by way of the hepatic vein the first changes occur in and about the central veins. In most cases, however, when the process is at all advanced it may be almost, if not quite, impossible to say from a single histological picture by which pathway the infection originated. Soon more marked degenerative changes occur in the hepatic parenchyma: the nuclei refuse to stain, the cells swell, hyperchromatosis, karyorrhexis, karyolysis, and plasmolysis ensue, until ultimately there is only an ill-defined mass of liquefaction necrosis, containing leukocytes, necrotic liver cells, bacteria, pigment, fat globules, detritus, etc., all of which is surrounded by a dense collection of leukocytes and fibroblastic infiltration that gradually fades away into the surrounding more or less normal liver tissue. Depending upon the pathway of infection, there are inflammatory and suppurative phenomena in the radicles of the portal vein, the hepatic artery, the hepatic vein, or the biliary ducts; not uncommonly the process originating in one of these sets of vessels spreads to the other, so that the composite picture much obscures the evidences of the primary infection.

The single, tropical, or amœbic abscess of the liver presents many characteristic features, which have been elsewhere described.¹

Symptoms.—Attempts are sometimes made to distinguish acute, subacute, and chronic abscesses; the distinction, however, is quite artificial, since although acute, subacute, and chronic abscesses do occur, a chronic abscess may remain latent for a long time and reveal itself acutely in consequence of some accidental disorder, such as trauma, intercurrent disease, etc., and suppuration that began acutely may subside and become virtually a chronic abscess. The symptoms of suppurative hepatitis vary with the nature and seat of the antecedent disorder, the pathway of infection, and the nature and virulence of the infectious agent. Many cases, especially of so-called tropical abscess, are entirely latent; often they are not detected until they have lasted some time; not uncommonly they constitute an unexpected finding at the necropsy (13 per cent. of the cases, Rouis).

Traumatic abscess as well as abscess due to spread of disease from adjacent structures, usually manifests itself by: pain in the region of the liver, aggravated by motion and pressure, and referred to the right shoulder; palpable and audible friction (attributable to local perihepatitis); perhaps jaundice due to compression of the biliary ducts; chills, fever, sweats, and leukocytosis; enlargement of the liver, usually irregular; and perhaps a fluctuating tumor. The sequence of these phenomena upon an obvious trauma, especially if the skin and subcutaneous tissues have been lacerated, gives to them peculiar diagnostic significance that can scarcely be misin-

¹ Consult Strong in this work, vol. i, 514.

terpreted; to these are sometimes added the discharge of pus through the wound, whereupon one has only to decide whether the abscess is about or involves the liver tissue. The detection of hepatic cells in the pus or in scrapings from the base of the abscess cavity settles the diagnosis conclusively, which, however, is usually a matter of academic interest, since the treatment cannot be other than surgical.

Multiple septicopyæmic abscesses, whether due to portal vein, hepatic artery, hepatic vein, or biliary duct infection, often are not diagnosed; in perhaps the majority of cases their existence can be only suspected; that is, the symptoms of the primary disorder overshadow those due to the infection of the liver and dominate the clinical picture. In the one case the symptoms are those of a primary suppurative process in the heart (ulcerative endocarditis, purulent pericarditis), in another case in the lungs (abscess of lung or empyema); again, in the abdominal or pelvic viscera or in the extremities. The primary local process may be more or less obvious, and the general symptoms, chills, fever, sweats, and leukocytosis, are usually referred thereto; pain in the region of the liver may be inconspicuous or misinterpreted; the liver may not be noticeably enlarged (since death usually ensues early); pain in the shoulder may be interpreted as that of septicopyæmic or toxic arthritis, etc. In many cases the diagnosis of the liver involvement is of academic rather than of practical importance, since the condition is one of general septicopyæmia, of which the hepatic involvement may be only a minor part; and its detection would not notably influence either the diagnosis or the treatment, although it might add materially to the seriousness of the prognosis. In some cases, however, particularly of so-called portal pyæmia, but also of general arterial pyæmia, or of spread of infection to the liver from suppurative cholangitis, the diagnosis is possible from a knowledge of the primary disorder and the development of local symptoms referable to the liver, the onset of which may be manifest by an aggravation of the general symptoms, especially the fever, sweating, and leukocytosis (or the marked lessening of a previous well-marked leukocytosis, significant of lessening general resistance). The significant local symptoms consist of pain in the region of the liver referred to the right shoulder, tenderness about the liver, perhaps a mild grade of jaundice, and enlargement of the liver; the enlargement may be quite regular, but the organ is likely to be softer rather than harder than normally; sometimes the enlargement is irregular and nodular, and the nodules may be soft or semifluctuating; in this event audible or palpable friction (attributable to perihepatitis) may be detected.

The so-called tropical abscesses and other liver abscesses of *intestinal origin* and slow (or comparatively slow) development are, as a rule, much more readily diagnosed; but, as already stated, some of them are so indefinite in their clinical manifestations as to escape detection. Generally the onset is slow and insidious; sometimes for weeks or months the patient may complain of general ill-health, malaise, or gradually increasing weakness—which are especially significant if they constitute part of the poor health or delayed convalescence from dysentery and other intestinal disorders attended by suppuration or ulceration (such as appendicitis, etc.). In some cases, however, the onset is sudden, attended by a chill or chilliness, and follows directly upon these intestinal diseases; in other cases, although the noteworthy symptoms develop acutely, the antecedent disorder was present at some more or less remote time, and the abscess, doubtless pre-

viously latent, has been awakened into activity by some accidental cause. In another series of cases there have been one or several, sometimes recurring, attacks of what one may interpret as acute congestion of the liver, or acute hepatitis, especially in the tropics.

Suppuration of the liver is sometimes first suggested by pain, but one must remember that even a very large abscess of the liver may be altogether unattended by pain. In some cases early in the disease the pain is like that already mentioned, in connection with active congestion or acute non-suppurative inflammation of the liver (due to tension of the capsule), which in reality usually precede the abscess formation. Pain when present varies with the situation of the abscess; when situated deep within the liver there may be no pain whatever, since the liver parenchyma is insensible to pain; the nearer the surface and the more marked the involvement of the liver capsule the more marked the pain; it may be quite acute, or merely a sense of weight or discomfort that may be increased by motion. With the progress of the abscess formation deep within the liver substance, the initial pain, that due to tension on the liver capsule, may entirely disappear (often occasioning an unwarranted sense of satisfaction); and it reappears when the abscess has reached the surface of the liver and provoked a perihepatitis, in which event the pain may be quite like that of pleuritis. The pain is not infrequently referred to the right shoulder, especially below the acromion process of the scapula, where it is usually dull and aching; this is said to be most common when the abscess is in the right lobe of the liver (the great majority of cases); occasionally it is referred to the left shoulder (abscess in the left lobe, in some cases at least); rarely it is bilateral. This pain also is due to the perihepatitis and diaphragmatis, which irritate the phrenic nerve which communicates with the superficial cervical plexus; the pain, therefore, is not significant of suppuration as such.

Jaundice is present in about 16 per cent. of the cases (58 of 375 cases, Thierfelder); usually it is slight, rarely marked. It is due to pressure of the abscess on adjacent biliary ducts or to an associated intrahepatic cholangitis.

Constitutional symptoms, especially chills, fever, and sweating, significant of suppuration under all circumstances, are of such importance that the diagnosis virtually is impossible in their absence. A chill is not uncommon at the onset of the suppuration, and a chill or chilliness, especially toward evening with a rise in the temperature, is rather frequent; but in some cases that develop slowly and become well encapsulated a chill may be missed—entirely or until late in the course of the disease. Fever is an extremely important symptom, and probably essential to the diagnosis. It varies much in different cases. In perhaps the majority of cases it partakes of the so-called hectic type, the elevation being in the evening; but the fever may be more or less continuous (especially early in the disease), remittent, or intermittent; sometimes the diurnal fluctuations are quite marked—96° to 104° or 105° F. In chronic and well-encapsulated cases the fever range may be slight; sometimes there is apparently no fever at all or only slight and transitory attacks; rarely the temperature is subnormal. Sweating is a common, almost a constant symptom; it is usually most marked at night, but may occur irregularly during the day, especially if the patient sleeps. Often the sweats are very profuse, and give rise to great debility. The skin in many cases is frequently moist and clammy, especially upon the slightest exertion. Other manifestations of the general toxæmia are seen in arthritic

pains, and sometimes swelling of the joints (toxic synovitis and arthritis), and in certain nervous symptoms, such as headache, nervousness, restlessness, insomnia, irritability, mental depression, melancholia, etc. These nervous symptoms may be due in part to the bacterial toxæmia and in part to gastrointestinal autotoxæmia, the consequence of interference with the detoxifying function of the liver.

Examination of the patient often discloses a general and facial aspect, the hepatic facies, readily recognized and of much diagnostic significance to those of experience. The patient obviously is sick; the facial aspect is one of anxiety, distress, and more or less suffering (or perhaps the marks of past suffering); the complexion is muddy or sallow, or slightly icteric; the general integument is now hot and dry, now cold and moist; loss of flesh is progressive and is more marked the more severe and the longer the duration of the disease.

Examination usually shows the liver to be enlarged, although in some cases when the abscess is deeply seated and small, and sometimes even when it is quite large, no very noteworthy enlargement may be detected by the ordinary methods of physical examination. The situation and direction of the enlargement depend upon the situation and direction of the abscess; since this is usually in the right lobe, the enlargement is usually in the right hypochondrium. In the early stages, when the abscess is still within the substance of the liver the enlargement is usually tolerably uniform; but as the abscess increases in size it commonly causes disproportionate enlargement of the liver upward and a corresponding change in the area of hepatic dullness; this often assumes a characteristic dome shape; that is, the upper limit of hepatic dullness rises near the mammillary line, reaches its highest point near the midaxillary line (where it may be on a level with the fourth or the third rib), and then descends, so that in the midscapular line it may be as low as the angle of the scapula. A decline of the upper limit of hepatic dullness near the vertebræ is quite characteristic of liver abscess. In some cases these changes are not present, as when the abscess occupies the left lobe, or when the enlargement of the liver is laterally rather than upward, or downward and anteriorly; in the last mentioned event there may be bulging beneath the margin of the ribs or in the epigastrium, where on palpation one may detect a rounded or globular, smooth, and tense or semi-fluctuating tumor (depending upon whether the abscess actually projects beyond the surface or is still within the substance of the liver). In some cases definite fluctuation may be elicited. When enlargement cannot be made out by the usual methods, recourse may be had to the *x*-rays, which will readily disclose any irregular enlargement as well as the usual limitation of the movement of the diaphragm on the right side in these cases.

As the abscess approaches or reaches the surface of the liver perihepatitis is set up, and friction may be detected; this suggests the point of election in the event of resorting to exploratory puncture. The muscles of the right upper quadrant of the abdomen become tense and unyielding, and there is usually considerable local tenderness. In some cases also there is more or less local œdema, confined to one or two intercostal spaces or involving almost if not quite all the surface projection of the liver; when present, this is of much diagnostic significance.

The urine is concentrated, of high color, increased specific gravity, and deposits an abundant sediment of urates and uric acid; as a rule, the amount

of urea is diminished, but this is quite variable, although it is likely to be lessened if there is much destruction of the liver tissue; indican and a small amount of albumin are sometimes present, and albumose may be found in cases of considerable toxic absorption. Examination of the blood may reveal leukocytosis—a total count of 25,000 or more—and a relative increase of the polynuclear neutrophiles; unfortunately, however, leukocytosis is not always found; it is likely to be absent in slow-growing, chronic, and well-encapsulated abscesses.

There are a number of other signs and symptoms that occur in varying combinations in different cases and are due to variable conditions. Thus, certain gastro-intestinal symptoms may develop, such as nausea and vomiting, which are most common when the left lobe is involved (local adhesions or pressure on the pylorus or duodenum); the appetite is usually poor, and the tongue coated in the centre, or smooth, red, and fissured; there may be constipation (usually when the abscess is latent) or diarrhoea (when there is marked congestion of the liver or the acute hepatitis of the tropics); sometimes an obsolete dysentery is reactivated; and large amounts of pus may appear in the stools if the abscess ruptures into the bowel. Amœbæ may be found in the stools, especially if there is diarrhoea; when there is constipation they may be found in the mucus that often coats the faeces. The growth of the abscess upward, together with restricted movement of the base of the right chest and of the diaphragm, cause more or less congestion and compression of the base of the right lung, and lead to cough, dyspnoea, impaired percussion resonance, feeble or bronchovesicular breath sounds, and fine rales; later, pneumonia may develop. In some cases a short, hacking, spasmodic cough ensues; it is usually referable to irritation of the diaphragm or an actual pleuritis, which in turn may have been due to transport of infection through the diaphragm or to rupture of the liver abscess into the pleura. The cardiac action also may be embarrassed by the pressure of a large abscess; and the pulse may be rapid and irregular. The spleen usually is not enlarged and not palpable. Ascites does not occur unless there is some causative complication.

Spontaneous *rupture* is a common event in the course of large, especially tropical, abscesses of the liver. In Cyr's series of 563 cases, rupture occurred in 159 (28.2 per cent.), as follows: Into the lung in 59, the pleura in 31, the pericardium in 1, the peritoneum in 39, the intestines in 13, the stomach in 8, the kidney in 2, etc. In Thierfelder's collection of 170 cases perforation occurred into the lung in 74, the pleura in 26, the pericardium in 4, the intestines in 32, the peritoneum in 23, the stomach in 13, the kidney in 1, etc. Additional statistics are given by Strong.

In a majority of the cases rupture occurs upward through the diaphragm (57 per cent. of Cyr's cases, 59 per cent. of Thierfelder's cases). In some cases purulent pleuritis results and gives rise to the usual signs thereof; in other cases (hepatopulmonary abscess) the pus finds its way into the lung either directly, in consequence of the early formation of adhesions (which is the rule), or indirectly, following a purulent pleuritis. When rupture occurs into the lung and the process finally involves and perforates a bronchus, a large amount of pus (perhaps mixed with blood) may be suddenly coughed up, or it may come up with very little effort, being evidently discharged in consequence of the release of the tension of the abscess; so great may be the amount of pus thus released that the patient's lungs may be

inundated and sudden death may ensue. In other cases smaller amounts of pus are coughed up from time to time, but the total amount in the twenty-four hours may be considerable—ten to twelve ounces or more. The expectorated pus may exhibit ordinary purulent characteristics and contain necrotic liver tissue; usually, however, in the early stages at least, there is more or less blood mixed with the pus; this expectoration has been likened to anchovy sauce. Amœbæ may be found in it, should they be the exciting cause. Should the condition progress favorably, which it does in many cases, the amount of pus expectorated gradually diminishes and ultimately ceases, and the patient recovers. In other cases the expectoration lessens or ceases, but the patient's general condition becomes worse, and chills, fever, and sweats recur; evidently drainage has become insufficient and pus accumulates. Again, a large amount of pus may be discharged, and again the expectoration may cease; such variation in the phenomena (alternate emptying and refilling) may recur for some time until recovery eventually ensues or the patient dies of septicopyæmia, rupture of a pulmonary blood-vessel, etc. A second unruptured abscess should be suspected in the event of persistence of the septic phenomena, despite evident free drainage.

Rupture into the stomach or the duodenum is usually followed by epigastric distress, nausea, vomiting of considerable pus, diminution in the size of the liver, and perhaps relief of the local symptoms referable to the liver. Rupture into the intestine is followed by diarrhœa and pus in the stools; the pus frequently is overlooked, especially when the rupture occurs into the small intestine. Perforation into the peritoneum may occur into preformed adhesions, whence a localized extrahepatic abscess may ensue, or into the general peritoneal cavity, in which event a generalized peritonitis may be set up and soon lead to death; but sometimes, in view of the fact that the abscess may be sterile, peritonitis may not immediately ensue and the patient, if operated upon, may recover.

Diagnosis.—The diagnosis of suppurative hepatitis is often attended with considerable difficulty. The most suggestive signs are the etiological factors, which vary with the source of infection, but which should be carefully studied; pain in the region of the liver, perhaps referred to the right shoulder; progressive enlargement and tenderness of the liver; slight jaundice, and chills, fever, sweats, and leukocytosis. These symptoms, however, are not always outspoken; the leukocytosis, for instance, may be slight, and the so-called tropical abscess may be latent for a long time, and when somewhat active may give rise to quite indefinite symptoms.

Manson¹ states: "Golden rules in tropical practice are to think of hepatic abscess in all cases of progressive deterioration of health; and to suspect liver abscess in all obscure abdominal cases associated with evening rise of temperature, and this particularly if there be enlargement of or pain in the liver, leukocytosis, and a history of dysentery—not necessarily recent dysentery. If doubt exists, there should be no hesitation in having early recourse to the aspirator to clear up the diagnosis." "The most common mistakes in diagnosis are: (1) Failure to recognize the presence of disease of any description, even when an enormous abscess may occupy the liver. (2) Misinterpretation of the significance and nature of a basic pneumonia—a condition so often accompanying suppurative hepatitis. (3) Attributing

¹ *Tropical Diseases*, 1907, 4th edit., 510.

the fever symptomatic of liver abscess to malaria. (4) Mistaking other diseases for abscess of the liver, and vice versa—for example, hepatitis of a non-suppurative nature, such as that attending malarial attacks; suppurative hepatitis before the formation of abscesses; syphilitic disease of the liver—softening gummata which are often attended with fever of hectic type; pyelephlebitis; suppurating hydatid; gallstone and inflammation of the gall-bladder; subphrenic abscess; abscess of the abdominal or thoracic wall; pleurisy; encysted empyema; pyelitis of the right kidney; pernicious anaemia; leukocythæmia; scurvy and similar blood diseases associated with enlargement of the liver; ulcerative endocarditis; kala-azar; Malta fever; trypanosomiasis. Any of these may be attended with fever of hectic type, increased area of hepatic percussion dulness, and pain in and about the liver."

Malaria may be excluded by a history of antecedent dysentery or other etiological factor; evening rise in temperature (that of malaria being more common during the day); disproportionate enlargement of the liver as compared with the spleen (the reverse being the case in malaria); absence of malarial parasites in the blood; polynuclear leukocytosis; and the non-response to quinine. Cholelithiasis and Charcot's biliary intermittent fever may be excluded by the absence of a history of cholecystitis, cholelithiasis, and biliary infection; the absence of attacks of fever, etc., separated by longer or shorter periods of apyrexia; the non-occurrence of increase in the jaundice after each attack; and the absence of serious impairment of the general health despite the long duration of the disorder.

Prognosis.—The small multiple abscesses of the liver, whatever their etiology, are almost invariably fatal, death usually resulting within one to three weeks. Many of the large single so-called tropical abscesses eventuate in recovery; their duration, however, is uncertain, since it is not possible always to say when they began; they are often latent for a long time. The mortality in unoperated cases has varied up to 80 per cent., but Cantlie states that now the figures "have fallen to between 20 and 30 per cent., and there is no doubt that with earlier operations the mortality will be still further reduced." The outlook after rupture into the lung and evacuation through a bronchus (hepatopulmonary abscess) is rather favorable (75 per cent. of recoveries, De Castro), although the condition may last for months; rupture into the hollow abdominal viscera or externally may also be followed by recovery; but rupture into the peritoneum, the pericardium, the abdominal bloodvessels, etc., is usually fatal. Recurrence of the abscess may follow apparent recovery, especially if the patient remains in or soon returns to the tropics.

Treatment.—The treatment of such forms of suppurative hepatitis as are amenable to treatment is essentially surgical. The treatment of the multiple septicopyæmic abscesses is that of septicopyæmia in general; the liver condition neither modifies nor influences appreciably the treatment, which should be stimulating and supporting, with such drainage or other surgical treatment of the original focus of disease as may be possible. No sufficiently satisfactory and dependable method of treatment by means of sera, bacterial emulsions, etc., is yet available, but doubtless the future will bring it.

The diagnosis of the large single abscess furnishes the indication for its evacuation; and even when the diagnosis of a single abscess cannot be made positively, although hepatic suppuration is obviously present, resort should

be had to operation. Manson, Cantlie, and others of much experience advocate evacuating the abscess by means of a trocar and cannula.¹ Cantlie has devised a special instrument, the use of which he believes obviates the only danger to be feared—that of puncturing the inferior vena cava or the trunk of the portal vein before it enters the substance of the liver. The opinion of those of much experience is entitled to respect, and the favorable results of the treatment commend it to professional confidence; but one cannot be unmindful of other dangers, such as severe hemorrhage, perforation of one of the hollow abdominal viscera, rupture of the abscess into the peritoneal cavity, etc., so that the preferable operative procedure consists of opening and draining the abscess under view. In the event of perforation of the abscess, immediate operation is indicated; operation is indicated also in the event of perforation into the lung, hollow abdominal viscera, etc., but it need not be undertaken immediately, since in many of these cases spontaneous recovery ensues; signs warranting delay consist of good general condition of the patient, minor septic or toxic phenomena (chills, fever, sweats, etc.), free discharge of pus, by the bowel or by expectoration, improvement in the local conditions, etc.

CHRONIC INTERSTITIAL HEPATITIS: THE CIRRHOSES OF THE LIVER.

Chronic interstitial hepatitis, comprising the so-called cirrheses of the liver, has been much and variously classified—etiologically, anatomically, and clinically.² The disorder was first described by Vesalius, but the term cirrhosis was first employed by Laennec to describe the yellowish (*κίρρος*, yellowish) "hobnails," which he regarded as new-growth.

Eliminating from consideration cases of: (1) So-called capsular cirrhosis (more properly called perihepatitis or hepatic capsulitis), in which the liver tissue may be not at all affected, except perhaps secondarily in consequence of compression or by slight ingrowth of new connective tissue from the capsule; (2) so-called cardiac cirrhosis or cardiac liver, which develops in consequence of long-standing passive congestion; (3) syphilis of the liver, in which the lesions are specific and peculiar to that infection; and (4) circumscribed or focal fibroses which follow limited inflammatory and degenerative processes, focal necroses, chronic obstruction of the biliary ducts (pericholangitis), etc.—there are two types of chronic diffuse disorder of the liver, attended by fibrosis, to which the term cirrhosis may be limited. One of these disorders is very common; it is usually due to the misuse of alcohol; it is characterized by moderate enlargement of the liver (which in advanced stages may become reduced in size), by phenomena of portal obstruction (notably hæmatemesis and ascites), and by the absence or unimportance of jaundice; and after the development of symptoms it usually runs a comparatively short course (Laennec's portal, alcoholic, atrophic, or multilobular cirrhosis). The second of these disorders is rare; it is of unknown etiology; it is characterized by marked and persistent enlargement of the liver and spleen, by chronic jaundice, by periodic attacks of abdominal pain

¹ The details of the procedure may be found in Manson, *loc. cit.*, and Cantlie, *International Clinics*, 1904, 14th series, ii, 93.

² Consult Edwards, *International Clinics*, 1902, twelfth series, ii, 92.

and fever, and by the absence of manifestations of portal obstruction (notably ascites); and it runs a comparatively long course (Hanot's, biliary, hypertrophic, or monolobular cirrhosis).

There can be no serious objection to the use of the terms Laennec's and Hanot's cirrhosis, aside from that inherent in eponymic terms. Alcoholic cirrhosis accurately describes most, but not all, cases of Laennec's cirrhosis; but alcohol perhaps sometimes gives rise also to Hanot's cirrhosis, although this is a disputed point, and the study of the etiological factors in each case of cirrhosis is important from many points of view, particularly the therapeutic. The terms atrophic and hypertrophic, to designate, respectively, a small and a large liver, are most ill-advised and have led to the most confusion and misapprehension. There has been much discussion as to whether or not there is a so-called hypertrophic stage of atrophic or Laennec's cirrhosis, and this so-called hypertrophic stage has been confounded with what is spoken of as hypertrophic or Hanot's cirrhosis. Aside from notable variations in size that may occur within a comparatively short time in a liver the seat of Laennec's cirrhosis, a small or so-called atrophic liver is often hyperplastic to a considerable degree, and a large or so-called hypertrophic liver always exhibits considerable atrophy of the hepatic parenchyma; and most cases of Laennec's cirrhosis result fatally while the liver is still larger than normally; that is, in many cases the so-called atrophic liver does not become atrophic (smaller than normally), but remains to the end, as it were, hypertrophic. The terms assuredly are inadequate to describe the conditions, and should be discontinued.

The arrangement and disposition of the new connective tissue in cirrhosis has led to the use of terms such as multilobular, monolobular, unilobular, interlobular, intralobular, pericellular, etc. These terms accurately describe the lesions with which they are connoted, but they take no account whatever of the changes in the parenchyma, which are of prime importance. Difficulties arise when one attempts to correlate these terms with definite disease entities; the disposition of the connective tissue suggested by the several terms often occurs together in the same case; clinically the terms are of no use whatever.

The preferable terms for the two main types of cirrhosis, of which there are subtypes, are portal and biliary; portal, because the etiological factor is perhaps always transmitted by the portal circulation, the new-formed connective tissue is especially conspicuous in and about the portal spaces in the liver, and the obtrusive symptoms are those of portal obstruction; biliary cirrhosis, because the essential lesion is a radicular cholangitis and the conspicuous clinical feature is jaundice, due to obstruction to the free flow of bile.

Two subtypes of biliary cirrhosis are sometimes differentiated: (1) The hypertrophic biliary cirrhosis of Hanot; and (2) an obstructive biliary cirrhosis—by which is meant a condition of pericholangitic fibrosis spreading to the adjacent liver lobules in obstruction of the extrahepatic biliary ducts. The disorder was first described by Wickham Legg, and later by Charcot and Gombault, Gilbert and Sourmont, Chauffard, Ford,¹ Weber,² etc. There is no doubt that fibrosis does occur in the liver in some cases of

¹ *American Journal of the Medical Sciences*, 1901, exxi, 60.

² *Transactions of the Pathological Society of London*, 1903, liv, 103.

chronic obstruction of the extrahepatic biliary ducts; but the fibrosis is usually minor in grade, it develops about the intrahepatic biliary ducts (pericholangitis), it seldom implicates the liver lobules to a noteworthy degree, and of itself it does not give rise to noteworthy symptoms; the symptoms remain those of the obstruction (especially chronic jaundice and acholic stools), to which subsequently may be added, as in any disorder of the liver, phenomena attributable to disorganization of the hepatic parenchyma. Compression of the common bile duct, with complete interruption to the flow of bile, as occurs, for instance, in carcinoma of the head of the pancreas, is often attended by serious consequences, such as dilatation of the intrahepatic ducts, and compression atrophy, degeneration, and pigmentation of the liver cells; in some cases also some replacement fibrosis occurs, but in degree it is by no means comparable to that of cirrhosis in the ordinary sense. The obvious inference is that in most cases of considerable fibrosis of the liver in chronic obstruction of the biliary ducts, factors other than the obstruction are the real cause of the fibrosis. In some cases this is doubtless infection of the biliary tract, with consecutive cholangitis and pericholangitis, as occurs in some cases of cholelithiasis, with more or less incomplete obstruction; in other cases, the common causes of portal cirrhosis, such as alcoholism, putrefactive and other disorders of the intestine (due in part perhaps to absence of bile), etc. The proper interpretation of the phenomena is of more importance and interest than attaches to academic discussions; those that interpret localized fibroses as cirrhosis will continue to speak of the condition as obstructive biliary cirrhosis; but it is not beyond the facts to assert that biliary obstruction of itself does not give rise to cirrhosis in an acceptable sense, that the fibrosis that does occur is not of constant type, is almost negligible anatomically, and is assuredly negligible clinically, and that when a noteworthy cirrhosis occurs in association with obstruction to the biliary ducts it is probably due to the ordinary causes of cirrhosis rather than to the obstruction.

PORTAL CIRRHOSIS.

Portal cirrhosis is a chronic degenerative and inflammatory disease of the liver characterized by recurring degeneration and regeneration of the hepatic parenchyma and by concomitant and consecutive fibrosis in and about the interlobular or portal spaces, all of which leads ultimately to obstruction of the portal circulation.

Etiology.—Portal cirrhosis is peculiarly a disease of adult life; it is most common during the fifth decade, the majority of the subjects succumbing before the fiftieth year, forty-eight and seven-tenths years being the average age of 121 males, and forty-seven years of 44 females, Rolleston. It is, however, by no means rare at the extremes of life. A notable number of cases occur in young children, in whom it is often fatal before the sixth year. It is a little more than twice as common in men as in women; and it is also more commonly latent in men. Occupation is of significance only in so much as it brings in its train other etiological factors, notably alcoholism.

Portal cirrhosis is undoubtedly the expression of the activity of some poison or poisons; these are various in nature, and may reach the liver by way of the portal vein, the hepatic artery, or both. In the great majority of

cases the portal vein is unquestionably the pathway. From time immemorial major importance has been attributed to alcohol, and assuredly with reason; but during recent years attempts have been made to minimize its influence, and its action is probably indirect rather than direct. Alcohol is especially active when taken in the form of distilled liquors, but cirrhosis occurs also in those addicted to the use of wine, beer, ale, porter, etc., and it is more common among those who drink constantly a small amount than among those who occasionally drink a considerable amount. The stronger liquors are the more active probably because they are often taken on an empty stomach and consequently reach the liver in concentrated form. How the alcohol acts is not definitely known, but it is likely that it acts: (1) As a direct irritant or poison to the liver cells (of which there is sufficient experimental proof), causing parenchymatous and fatty degeneration, even necrosis, or disturbing the functions of the liver cells and rendering them unduly susceptible to the influence of other causative factors (auto-genic poisons, bacterial toxins, etc.), as well as irritating the connective tissue of the organ; and (2) by causing gastro-intestinal catarrh and thus favoring the formation of divers autotoxins inseparable from that condition which, transported by the portal vein, may be the active factor in provoking cirrhosis in a liver otherwise rendered vulnerable. It has been suggested that ethyl alcohol is not the active factor in setting up the cirrhosis, but rather adulterants, such as amyl alcohol, the aldehydes, aromatic substances, etc.; but as yet this is pure hypothesis. Certainly, however, cirrhosis occurs in the absence of alcoholism, and not all alcoholics become cirrhotic. In cases in which alcohol seems to be, or is, the only obviously active factor, it is not unlikely that there is also a concealed hereditary or a postnatal acquired lowered vitality on the part of the liver that renders it especially susceptible; and this is doubtless true also in respect to other etiological factors. That hereditary influence is sometimes operative is suggested by the occasional occurrence of several cases of cirrhosis in the one family; but in these cases one cannot overlook the likelihood of familial addiction to alcoholism, as well as hereditary syphilis.

There is reason to believe that poisons, particularly the fatty acids (lactic, butyric, acetic, valerianic, etc.) and other ill-understood enterogenic toxins associated with different forms of indigestion, may be the causative factors, especially in non-alcoholic subjects who give a history of long-continued indigestion, following or associated with overindulgence in highly seasoned foods, spices, etc. (dyspeptic cirrhosis, Budd's non-alcoholic cirrhosis). The fatty acids and other toxins rarely may be ingested preformed with the food; but they are much more likely to be manufactured in the intestinal tract in cases of gastro-intestinal catarrh (fermentative dyspepsia); experimentally lesions resembling those of cirrhosis have been produced by injecting fatty acids into lower animals. To the influence of enterogenic toxins, spices, etc., may also be attributed the cirrhosis occurring among the Hindus, Egyptians, etc., who, avoiding alcohol, nevertheless often eat food that is not above reproach and are quite partial to condiments and spices, such as ginger, pepper, etc., often in decoctions. It is also likely that a poison elaborated in the spleen may cause cirrhosis (Banti's disease, splenic anæmia with terminal cirrhosis of the liver, and ascites).

Infections of various kinds may be followed by cirrhosis, but the relation-ship of the one to the other has not yet been definitely determined. In most

of these cases the infection is carried to the liver by the general as well as by the portal circulation. Many acute infections are known to cause pathological changes in the liver (degeneration, focal necroses, inflammation, etc.), and some of these are followed by fibrosis—usually localized and minor or moderate in grade; but this scarcely constitutes cirrhosis in an acceptable sense. A malarial cirrhosis has been widely commented upon, but it is probably not as common as many observers believe; both diseases may occur concurrently. Tuberculosis is sometimes associated with cirrhosis, especially fatty cirrhosis, and some observers believe that the tubercle bacillus or the tubercle toxin absorbed from the intestinal tract or the peritoneum may set up the lesions of cirrhosis.¹ The lesions in the liver caused by syphilis are not those of cirrhosis (in a restricted sense), but antecedent syphilis may render a liver unusually susceptible to the ordinary causes of cirrhosis. A form of cirrhosis occurring in Egypt and elsewhere has been attributed to a toxin elaborated by *Uncinaria duodenalis* and *Bilharzia hæmatobia*, with which these subjects are infected. Rogers² has described a peculiar form of intralobular cirrhosis produced by the protozoal parasite of Kala-azar. Adami's observations relating to a so-called diplococcal form of the colon bacillus in cirrhosis in man and in infective cirrhosis in cattle is of interest, not so much as suggesting an etiological relationship of the bacterium to cirrhosis, but as illustrating the bactericidal properties of the liver cells. It is not improbable that bacteria may be indirectly the cause of cirrhosis in setting up catarrhal and fermentative processes in the intestine, the toxic concomitants of which, being transported to the liver, may act as the direct irritant factor.

Other sorts of irritants sometimes appear to give rise to cirrhosis. Thus in hæmochromatosis, in consequence of hæmolysis, blood pigment is set free and infiltrates the tissues, and the liver and the pancreas become fibrotic. Cirrhosis of the liver has been found in association with pneumokoniosis (anthracosis, etc.), and with poisoning with lead, silver, arsenic, etc.

Experimentally,³ so-called cirrhosis, but usually minor and circumscribed fibrosis, has been produced by injecting certain bacteria, alcohol, and other irritants, such as chemicals, vegetable alkaloids, etc., into the portal circulation, the general circulation, and the intestine. Much more significant and of more importance are Pearce's⁴ studies regarding the effects of hæmolytic and hæmagglutinative sera: the early necrotic lesions in the liver are followed by reparative processes that constitute a chronic interstitial hepatitis; these are of special interest, since they demonstrate that cirrhosis may follow widespread destruction of necrotic lesions in the liver, and explain the histogenesis of cirrhosis and other reparative processes in the liver.

Pathology.—The liver varies much in size in different cases, and in the same case at different times. At the necropsy it may be very small, weighing only 980 grams or less; or it may be very large, weighing as much as 4000 grams or more; but it is worthy of note that in the majority of cases at the necropsy it weighs more than normally. Rolleston states that the average

¹ Consult Jagie, *Wiener klinische Wochenschrift*, 1907, xx, 849, Stoerk, *ibid.*, 1907, xx, 1011, 1048; and Isaac, *Frankfurter Zeitschrift für Pathologie*, 1908, ii, 1250.

² *Annals of Tropical Medicine and Parasitology*, July, 1908.

³ Consult Fischler, *Deutsches Archiv für klinische Medizin*, 1908, xciii, 427.

⁴ *Journal of Experimental Medicine*, 1906, viii, 64; *Journal of Medical Research*, 1906, xv, 99 (literature).

weight of the liver in 155 consecutive necropsies on cirrhotic subjects at St. George's Hospital was 63.6 ounces (1978 grams); the average weight of the liver in 100 cases collected by Hawkins was 52 ounces (1617 grams), the minimum being 32 ounces (995 grams), and the maximum 74 ounces (2300 grams); the average weight in 93 cases collected by Kelynack was 53 ounces (1648 grams); and the average weight in 34 cases at the German Hospital, Philadelphia was 1875 grams, the minimum being 980 grams and the maximum 2760 grams. In fatal cases in young subjects the liver is relatively and often absolutely larger than in older subjects.

Variations in the size of the liver may be due to different factors. It is essential to bear in mind that in the usual course of events the liver may be enlarged from the beginning to the end of the disease. It is often said that the liver is enlarged in the early stages of the disease and small in the later stages—in consequence of contraction of the newly formed fibrous tissue. This change in size is sometimes observed, but it is incorrect to assume that the decrease in size is due always or solely to contraction of the new fibrous tissue. Marked variations in size often occur rapidly, within several days or weeks, and are due solely to changes in the vascular supply. The writer saw a liver a hand's breadth below the costal margin in the nipple line lessen so rapidly in size that at operation two weeks later it had receded well above the costal margin and presented the typical appearances of portal cirrhosis. Similarly, large livers may be considerably reduced in size at the necropsy several weeks later; and livers small at one clinical examination may be much enlarged subsequently. The large liver may be due to vascular engorgement, hyperplasia of the liver cells, disproportionate fibrosis that is likely to be diffuse, as in the so-called monolobular type, and fatty infiltration and degeneration of the liver cells (fatty cirrhosis), often attributed to the influence of malt liquors and frequently associated with pulmonary tuberculosis.

As a rule, the liver is of increased density (increased specific gravity), so that a small liver may weigh more than a liver of normal size; it is of increased consistency and lessened elasticity. The capsule is often opaque, sometimes considerably thickened, and not infrequently adherent to the under surface of the diaphragm. Adhesions, when present, are usually old, fibrous, and circumscribed rather than widespread; occasionally, however, they are almost if not quite universal and may extend to adjacent organs—the gall-bladder, stomach, intestine, omentum, through the diaphragm to the pleura, lung, etc. In other cases more or less recent, acute, fibrinous adhesions are encountered.

The surface of the liver is distinctly granular, the small livers more so than the larger ones: grayish-white opaque depressions alternate with pale, yellowish-brown, sometimes reddish-brown, roundish or ovoid elevations—the so-called granular or "hobnail" liver. The hobnails vary considerably in size, from those scarcely larger than a pin's head (under which circumstances the surface of the liver is comparatively smooth) to others 1 to 2 cm. in diameter; usually they average from 2 to 4 mm. in diameter. Sometimes many—ten or more—hobnails appear especially surrounded by an unusually wide band of connective tissue that isolates them from adjacent tissue and causes a more marked projection from the general surface of the organ. These lesions usually are distributed uniformly

throughout the liver, but occasionally one lobe is more involved than the others; the left lobe especially may be very small (lessened resistance?); rarely the caudate, quadrate, or Spigelian lobe may be disproportionately affected.

On section the liver cuts with increased resistance, being much denser, firmer, and tougher than normally. The cut surface reveals interlacing bands of whitish or grayish-white fibrous connective tissue that vary somewhat in thickness, pervade the entire organ, and are continuous with the grayish opaque depressions on the surface of the liver. These bands of fibrous connective tissue, which obviously take their origin in the periportal spaces, form a network that encloses or surrounds islets of liver tissue that vary in size from that of a pinpoint to others 1 cm. or more in diameter; the larger islets are made up of several, often eight, ten, or more, liver lobules (or remains of liver lobules), and are pervaded by more delicate bands of connective tissue (so-called multilobular cirrhosis). In many, but not all, cases the islets of liver cells appear to be under some tension, since on section they project somewhat; it is not improbable that this is due to the elastic tissue of which in part the newly formed connective tissue consists. The liver tissue is usually pale yellowish in color or yellowish brown; occasionally it is greenish (from staining with bile), or reddish or brownish, especially in the centres of the lobules (from staining with blood or blood pigment). The islets of liver tissue are sometimes very large, hyperplastic, evidently the result of active proliferation, a condition sometimes spoken of as nodular cirrhosis, or cirrhosis with multiple adenoma. This process doubtless, at first compensatory, sometimes becomes unrestrained and results in the production of carcinoma (cirrhosis with carcinoma). Occasionally the islets of liver tissue are very pale (fatty degeneration) and at first sight resemble new-growth; in some cases the centres of some of the nodules are quite soft and necrotic.

The bloodvessels of the liver show more or less well-marked changes. The hepatic artery and its smaller branches are often distended—doubtless related etiologically to the necessity of supplying with blood the increased fibrous tissue; in some cases the vessels exhibit the ordinary lesions of endarteritis, especially in the cirrhosis of hæmochromatosis and of syphilis. The hepatic veins may show the lesions of obliterating endophlebitis, as has been pointed out by Hess.¹ The smaller intrahepatic branches of the portal vein, especially those between the liver lobules, are often compressed and not infrequently thrombosed; the larger intrahepatic branches, the main trunk, and the gastro-intestinal branches that go to form the main trunk are usually dilated; the wall of the vein is often thickened—periphlebitis and endophlebitis; and thrombosis of the main trunk may occur (more common in cirrhosis than in any other condition).

Communications between the portal vein and the systemic circulation constitute a part of the cirrhotic process. For the most part these consist of dilatations of normally existing anastomoses. They subserve the useful purpose of diverting to the superior and the inferior venæ cavæ the venous blood in the portal tributaries flowing against the obstruction in the portal distribution in the liver, and thus they relieve the portal congestion. Although usually present, they are not always observed; they are sometimes absent in

¹ *American Journal of the Medical Sciences*, 1905, cxxx, 936.

fatal cases of cirrhosis in which death has resulted from other causes. These anastomoses have been thus described by Rolleston:¹

"1. A general anastomosis between the veins of the peritoneum and those of the abdominal walls, such as the lumbar and renal. These anastomoses are especially well-marked where the duodenum and colon are bound down to the abdominal wall and are only partially covered by peritoneum. This subperitoneal anastomosis, described by Retzius, gives rise to marked injection of the peritoneum, which is especially noticeable during life, as seen in laparotomies on cases of cirrhosis.

"2. Those around or in connection with the liver. The phrenic and intercostal veins on the diaphragm communicate between the layers of the coronary ligament with the veins in the liver; this is not of much utility. Dendritic nervous markings on the skin along the line of attachment of the diaphragm occur in conditions like emphysema, and have no constant relation to cirrhosis. In the falciform ligament the parumbilical veins of Sappey put the portal vein into communication with the veins of the anterior abdominal wall. A large vein may thus run up in the falciform ligament which imitates the anterior epigastric vein of the frog. This anastomosis may show itself as a 'caput medusæ,' or number of dilated veins around the umbilicus. This anastomosis must be distinguished from the more marked 'caput medusæ' which results from obstruction to the passage of blood along the inferior vena cava; in the latter the dilated epigastric and mammary veins avoid and do not centre around the umbilicus. In cases of extensive ascites, both collateral circulatory channels may be developed.

"3. Between the œsophageal veins, discharging onto the azygos veins and so into the superior vena cava on the one hand and the gastric veins on the other hand. These veins may become varicose (œsophageal piles), and, as a result of chronic inflammation and mucous membrane, may become first adherent and then ulcerated. Profuse and even fatal hæmatemesis may thus be induced. In 80 per cent. of the cases of fatal gastro-intestinal hemorrhage these œsophageal varices are present. Varicose gastric veins, especially around the cardiac orifice, are present in a small proportion of cases.

"4. Between the superior hemorrhoidal veins, tributaries of the inferior mesenteric vein, and the middle and inferior hemorrhoidal veins, which open into the internal iliac veins. Dilatation and varicosity of these veins lead to piles. It is probable that cirrhosis is not so important a cause of piles as has sometimes been stated, and at any rate takes a very subordinate position in this respect to constipation."

These anastomoses serve both a useful and an evil purpose. On the one hand, as long as the collateral circulation is efficient obstructive symptoms are in abeyance and the disordered liver is enabled better to perform its functions, perhaps enabled to regenerate, whence in some cases the disease remains or becomes latent, even apparently cured; on the other hand (*a*) the dilated veins are likely to rupture, either spontaneously or in consequence of trauma, and lead to more or less severe hemorrhage (hæmatemesis, enter-rhagia, hæmatoma of the abdominal wall about the round ligament); and (*b*) much of the blood from the gastro-intestinal tract being thus diverted from the liver to the general circulation, conditions somewhat analogous to

¹ For an excellent study with a review of the literature consult also Gilbert and Villaret, *Revue de médecine*, 1907, xxvii, 305.

those induced by the establishment of an Eck's fistula (the experimental communication between the inferior vena cava and the portal vein), supervene, much of the products of digestion is not brought under the influence of the liver, is not detoxified, and toxæmia ensues.

The gall-bladder and the biliary ducts are usually normal; occasionally there is some thickening of the wall of the gall-bladder from chronic cholecystitis. Calculi are sometimes encountered in the gall-bladder or the ducts: in 21 (15.4 per cent.) of 136 fatal cases (Rolleston); in 18 (8.6 per cent.) of 209 men, and in 7 (17 per cent.) of 41 women (Klopstock); in 8 (22.2 per cent.) of 36 cases at the German Hospital, Philadelphia.

*Microscopy.*¹ A clear conception of the cirrhotic process is best attained by bearing in mind that, as recognized years ago by Hyrtl and more recently emphasized by Kretz,² the liver is not made up of acini in an acceptable sense. While the physiological unit of the liver is that collection of cells drained by a radicle of the bile duct, this unit is scarcely delimitable anatomically; nor are the so-called acini, or lobules, cross-sections of which, surrounded by connective tissues, form such a conspicuous feature of microscopic sections, of such simple structure as at first sight appears. Study of the liver in serial sections and its reconstruction show that the liver parenchyma does not form isolated acini, or lobules, everywhere surrounded by connective tissue, but that what appear to be acini are merely cross-sections of liver cells arranged as a mantle about the numerous dendritic ramifications of the hepatic vein, and that this mantle is nowhere discontinuous, forming, on the contrary, a continuous whole, everywhere connected, particularly at the points of junction of the sublobular, lobular, and lobar veins. The intimate relationship that the liver cells structurally bear to the hepatic vein is further shown by the radial disposition of the cells about the branches of this vein—the so-called central veins.

As distinguished from most other organs, the liver has two sources of blood supply—the portal vein and the hepatic artery—which commingling form a plexiform capillary network, in the meshes of which the liver cells are arranged. Ultimately, the capillaries unite again to form the radicles of the hepatic vein—the rectilinear veins of Sabourin which empty into the so-called central veins. The circulation within the liver, which is of much importance in connection with certain features of cirrhosis, has been studied attentively by Opie.³ It has long been known, and Opie especially has directed attention to the fact that one of the important characteristics of the liver is its susceptibility to variations in blood pressure, saying: "The portal vein, hepatic artery, and hepatic vein are in such free communication that the entire lobule can be injected from any one of these vessels. Hence artificial injections do not define the intralobular distribution of the portal and arterial blood, for doubtless differences of venous and arterial pressure have a part in determining peculiarities of the circulation within the lobule." Opie's experimental studies led him to the conclusion "that both the hepatic artery and

¹ This is largely an abstract of an article by A. O. J. Kelly, entitled the Nature and the Lesions of Cirrhosis of the Liver, with Special Reference to the Regeneration and Re-arrangement of the Liver Parenchyma, *American Journal of the Medical Sciences*, 1905, cxxx, 951.

² Ueber Lebercirrhose, *Wien. klin. Woch.*, 1900, xiii, 271; *Verhandl. der Deut. path. Gesellschaft*, 1904, viii, 54; *International Clinics*, 1905, iii, 289.

³ *Journal of Medical Research*, 1904, xii, 147.

portal vein pour their blood into the periphery of the lobule, and here the influence of the arterial blood pressure is most strongly felt. When foreign material reaches the liver by the portal vein it is washed from the peripheral zone by the arterial blood, and tends to be deposited in a middle zone, where the influence of the opposing circulation is less strongly felt. Hence it is not improbable that toxic substances capable of causing necrosis of the hepatic cells, brought to the liver by the portal vein, might first exert their effect upon cells within a midlobular zone. The periphery of the lobule, moreover, is supplied with arterial blood, and is perhaps for this reason less susceptible to injurious agencies."

If the parenchyma of the liver in a moderately advanced or well-advanced case of cirrhosis is studied attentively, certain noteworthy deviations from the normal become apparent: Changes in the size, shape, and configuration of the lobules, and in the arrangement of the liver cells; degenerative, atrophic, and hyperplastic changes in the liver cells; and changes in the vascular arrangement and supply.

The size of the lobules or "hobnails" in cirrhosis has been studied by Kretz,¹ MacCallum,² etc. Kretz, pointing out that the normal lobules are roundish polygonal in shape, with their long axis in the direction of the central vein, states (as is well known) that their greatest diameter is usually a little more than 1.5 mm., and may reach 2.5 mm., whereas the short diameter is about 1 mm., and that in cirrhosis these figures are surpassed, some lobules being much larger, and other collections of cells much smaller. MacCallum found that while the average radius of a lobule (that is, the thickness of the mantle of cells about the central vein) in a number of normal livers is about 0.66 mm., in advanced cirrhosis lobules are encountered that measure from central vein to periphery 1 mm. or more, the small diameter, therefore, being more than 2 mm. Personal observations amply confirm the truth of these statements.

There is a notable departure from the normal shape and configuration of the lobules; many of them are no longer rounded, but quite irregular in transverse section; many of the cell mantles no longer surround the central veins equally on all sides, but very unequally, so that many central veins are situated eccentrically in a more or less misshapen lobule; many so-called lobules have no central vein at all (and are not tangential sections of otherwise normally arranged lobules); certain lobules (or so-called lobules, usually remains of several) have two, three, four, or more central veins; and often the central vein may be seen in the adjacent interlobular connective tissue. Thus the mantle of cells covering a central vein varies much in thickness in different regions (transverse planes), sometimes exposing, sometimes covering, the vein to a greater or less extent; and although isolated collections of liver cells (that is, collections devoid of central veins) do occur, some supposedly isolated collections are merely sections of more or less irregularly proliferating dendritic branches of what may be called the parent stem, or mantle covering a single central vein.

Furthermore, although the normal radial arrangement of the liver cells about the central vein is preserved to some extent, in many places it is totally wanting; the architecture of the lobule has become altered so that the cells are arranged irregularly, in parallel rows, etc. This re-arrangement of

¹ *Loc. cit.*

² *Journal of the American Medical Association*, 1904, xliii, 649.

the liver cells is quite as well-marked in many of the lobules that have a central vein as in those collections of cells entirely devoid of central veins.

The parenchymatous cells show various degenerative changes. In the early stages many cells become degenerated, necrotic, and are removed, but from the nature and the stage of the process at which this occurs, it is rarely observed, except in cases that run a rapid course and in certain cases of pigmented cirrhosis. In some cases there is considerable or widespread fatty infiltration (so-called fatty cirrhosis). In advanced cases considerable atrophy of the liver cells may be observed. It is important to note that many cells at the periphery of lobules (or so-called "hobnails") in immediate contiguity to wide bands of connective tissue are by no means atrophic, even in fairly well-advanced cases; on the contrary, whereas a priori, compression atrophy of these cells should be expected, and is, indeed, often commented upon, the cells that should exhibit this alteration most markedly—that is, those of the periphery of the lobule—often present an unusually plump and healthy appearance, and cannot therefore have been unduly compressed.

Evidences of hyperplasia of the liver cells are readily found in almost every cirrhotic liver, even in far-advanced cases. Mitoses, it is true, are rarely encountered in advanced cases—largely in consequence of the nature of the process, the advanced stage at which the examination is generally undertaken, and the length of time after death that the liver is obtained, time sufficient for the completion of any mitoses present at death. That many of the liver cells, however, are newly formed is quite apparent from their large, clear, and plump appearance, their rounded (rather than polygonal) outline, the presence of two or more nuclei, and the absence of fat and of pigment from their protoplasm. These cells, sometimes small collections of them, are situated amidst other liver cells normal in size and outline, provided with only a single nucleus each (though sometimes with two nuclei, which is quite normal), and the protoplasm of which is normally pigmented and contains fat. These newly formed cells are usually found at the periphery of a lobule; sometimes they make up a larger or smaller sector of a lobule; sometimes they seem to have compressed the older cells in their immediate vicinity; and sometimes small collections of them are found completely surrounded by wide bands of connective tissue. The sector of a lobule sometimes made up of these cells serves, in some cases, to restore more or less completely the normal outline of the lobule, that is, of the mantle of cells covering a central vein—in which event a part of the mantle of cells consists of such newly formed cells arranged irregularly, and a part of older cells arranged radially; in other cases this cellular hyperplasia has occurred in such a manner that the central vein is more or less exposed on one side, and on the other covered with a mantle of cells often twice or thrice the normal thickness.

In acute and subacute, or what may be called recurring, cases mitoses are occasionally encountered (doubtless they would be more frequently encountered were our methods of investigation better thereto adapted), and necrotic and hyperplastic changes may be found side by side in the one lobule.

A characteristic feature of almost all cases of cirrhosis, more conspicuous, however, in some cases than in others, is the bile-ducts or so-called pseudo-biliary canaliculi, the nature and origin of which has for a long time occasioned much discussion. Doubtless the great increase of these structures in

cirrhosis, as contrasted with the normal liver, is relative only, and due in part, but only in small part, to mutual approximation of already existing bile ducts occurring in consequence of loss of the liver tissue; but that most of them are newly formed is generally conceded, and is quite apparent from their excessive number and the evidences of active proliferation (mitosis) that they commonly exhibit. Their origin has not been determined definitely for all cases. It seems quite certain that some, at least, of them result from proliferation of previously existing bile ducts; on the other hand, since they are found in a wide variety of disorders, such as all forms of cirrhosis, acute yellow atrophy, in the neighborhood of hydatid cysts, tubercles, gummas, etc., in other words, in diseases alike only in that they destroy liver tissue; since they differ from normal bile ducts in the absence or relative paucity of encircling elastic tissue; since they have been found by Ackermann and others to be directly continuous with liver cells; and since in cases in which the liver cells have been much destroyed, they are found in large numbers in what may be interpreted as the remains of the skeleton of the lobule, we are quite warranted in the opinion that they may result from proliferation of the liver cells themselves, constituting in this event a reversion to a less highly specialized type of structure. The important fact to point out is the histogenetic equivalence of the liver cells and the epithelium of the bile ducts, and the fact that proliferative changes of the liver parenchyma are a conspicuous feature of common cirrhosis.

Reference has already been made to the fact that the central veins are situated eccentrically in certain more or less distorted and re-arranged lobules; that they may be found at the edge of certain lobules or remains of lobules, and even in the interlobular connective tissue, and that certain islets of liver tissue are totally devoid of central veins. Another noteworthy feature is that the central veins appear to have diminished in number in the liver as a whole. Corresponding to the smaller size of many of the cirrhotic livers and the associated loss of the liver parenchyma, the central veins in certain regions sometimes exhibit a mutual approximation, which, however, varies much in different livers and in the same liver in different places. In some regions where considerable hepatic-cell hyperplasia has occurred they are often quite widely separated. But were the central veins not diminished in number in the liver as a whole, we should expect to find, in a small so-called atrophic cirrhotic liver a relative increase in the number of central veins in a given square surface as compared with a square surface of similar size in a normal liver—which, however, is not the case. The absolute lessening of the number of central veins becomes further evident when we recall the many islets of liver tissue (likely at first sight to be interpreted as more or less distorted lobules) entirely devoid of central veins.

Propos of the circulation in cirrhosis, Kratz states: "If in ordinary atrophic cirrhosis an injection is made from the portal vein a small part of the hepatic tissue is, as a rule, so easily permeated that the injection mass passes over into the hepatic vein at a time when a very considerable part of the parenchyma is still uninjected; more particularly after such injection experiments the large turgescient islets of tissue and the small areas devoid of hepatic veins . . . remain unaffected. By injecting the hepatic artery, always decidedly enlarged in these cases, these very areas are most easily injected. They may also be partly injected from the portal vein if an injection mass, which passes with difficulty, is employed. Based upon these observa-

tions, the circulation in the diseased liver must be depicted as follows: a small part of the portal blood passes through the remnants of the old acinous channels into the hepatic veins; this passage forms an internal portocaval anastomosis supplementing the external; to the remaining branches of the portal vein more than the normal amount of arterial blood is furnished; this, on the one hand, supplies the granular areas of the parenchyma devoid of hepatic veins, and on the other, the hypertrophic remains of acinous tissue. This excess of blood passes under increased pressure through the remainder of the capillaries, narrowed by atrophy of the acini and lengthened by hypertrophy of the liver tissue. The lengthening and narrowing of the capillary vessels and the excess of arterial blood are the true causes of the increase of portal pressure, as is shown by the occurrence of ascites in subacute atrophy of the liver with regeneration, but without contracting connective tissue."

The changes in the connective tissue vary with the stage and activity of the process. Early there is more or less round-cell infiltration in and about the portal spaces; later there is considerable increase in the amount of connective tissue—in part the result of organization of the inflammatory exudate, in part a replacement fibrosis to fill up potential lacunæ left by destroyed liver parenchyma. This newly formed connective tissue commonly encloses rather than invades the liver lobules; sometimes only one lobule is enclosed in a wide band of connective tissue, sometimes many lobules—whence the designation multilobular cirrhosis. In many regions, however, although the general distribution of the connective tissue is the so-called multilobular, there is more or less invasion of the lobules from the periphery, so that there is an approach to the type spoken of as monolobular, intralobular, or intercellular cirrhosis. This has led some observers to postulate "mixed types," which is unnecessary, since this distribution and arrangement of the connective tissue is quite common in portal cirrhosis, especially in the more active cases. In the active cases¹ there is always considerable round-cell infiltration and many newly formed and dilated capillaries and arterioles derived from the hepatic artery; in the less active and older cases both of these are less conspicuous, and the connective tissue partakes of the characteristics of a cicatrix. The occurrence and significance of lymphangitis and perilymphangitis have recently been commented on by Oertel.²

This new connective tissue consists of several sorts of tissue: Some of it is unquestionably newly formed, some of it is the tissue originally between the lobules—the connective tissue of Glisson's capsule, and some of it is the collapsed and otherwise altered reticulum of certain liver lobules the cells of which have disappeared. Whereas, formerly the fibrosis of cirrhosis was generally thought to consist almost, if not quite, exclusively of white fibrous tissue, the studies of Hohenemser, Melnikow-Raswedenko, Flexner, McIntyre, Oliver, and others have demonstrated that in reality it consists of all the connective tissues of the normal liver—pathologically altered, it is true. The normal liver contains three kinds of connective tissue: (1) White fibrous tissue, present in the liver capsule and in large amount in the interlobular spaces; (2) elastic tissue, found chiefly about the bloodvessels, to a less extent about the bile ducts and in the liver capsule, and to a very slight extent in and about the walls of the central veins; and (3) reticulum.

¹ Consult Symmers, *American Journal of the Medical Sciences*, 1908, cxxxv, 251.

² *Archives of Internal Medicine*, 1908, 1, 385.

A conspicuous change in cirrhosis is the great increase in the elastic tissue—probably more or less intimately related to the great increase in the capillaries derived from arterioles; this elastic tissue not only makes up a large part of the fibrous bands between the lobules, but it is also found to some extent within the lobules, and occasionally a very small amount of it is found about or in the immediate neighborhood of the central veins. A not inconsiderable part of the fibrosis is made up of reticulum—not only the reticulum normally between the lobules, but also the intralobular reticulum—that remaining after the liver cells have degenerated and been removed. This collapsing intralobular reticulum may be quite readily distinguished in tolerably early cases; later, however, when it has become a part of the interlobular fibrosis it can scarcely be recognized as a distinct entity. Flexner is quite correct in the opinion that the changes in the reticulum in cirrhosis constitute a hypertrophy rather than a hyperplasia of the fibrils.

Lesions in Organs other than the Liver.—The spleen is enlarged in at least 80 per cent. of the cases (198 of 250 cases, Klapstock;¹) it is probably enlarged sometime during the course of all cases, but variations in size are due to different factors. There is no constant relationship between the size of the liver and of the spleen. At the necropsy the average weight of the spleen is about 400 grams; the average weight of 32 cases at the German Hospital, Philadelphia, was 400 grams, the minimum 110 grams, and the maximum 1320 grams. In the early stages, in consequence of increased vascularity, proliferation of the splenic pulp, and hyperplasia of the endothelial cells of the sinuses, the organ is about normal in consistency; later it becomes firmer and harder, in consequence of increase of the trabeculae and consecutive atrophy of the lymphoid elements, particularly the Malpighian bodies. Sometimes, however, late in the disease more or less extensive softening may be encountered—due to hemorrhage or terminal infections. Considerable and quite rapid diminution in the size of the spleen may follow gastro-intestinal hemorrhage, severe diarrhoea, the recurrence of ascites after tapping, etc. Chronic perisplenitis occurs in about one-third of the cases; it may be localized or generalized. In consequence thereof, the spleen is usually adherent to the adjacent organs—the diaphragm, the abdominal wall, the stomach, the colon, etc.

The enlargement of the spleen is doubtless due to two factors: (1) Toxæmia, bacterial or other toxins brought to the organ by the general circulation—which is especially operative during the early stages, since enlargement is not frequently observed early, before there is any evidence of portal obstruction; and (2) passive congestion—which is especially operative, although not the sole factor, in the later stages; undoubtedly it serves mechanically to distend the blood spaces, and, obstructing the return circulation, it serves to retain irritating poisons in the organ.

The gastro-intestinal tract shows lesions due in part to the cause of the cirrhosis (alcoholism especially), in part to portal obstruction. The œsophagus shows general congestion, thickening of the mucosa, and in 80 per cent. of the cases, dilated and tortuous veins, especially toward the lower end; these not infrequently rupture and give rise to severe and even fatal hemorrhage. The stomach is the seat of chronic gastritis, often associated with small superficial ulcers or abrasions; pigmentary infiltration, especially

¹ *Virchow's Archiv*, 1907, clxxxvii, 111.

toward the pylorus; and dilated veins, especially toward the cardia; these also frequently rupture. The intestines reveal the ordinary lesions of chronic passive congestion and chronic enteritis with pigmentation. The rectum is often congested and the seat of small superficial ulcers and of dilated veins (hemorrhoids), both of which may lead to more or less severe hemorrhage. The peritoneum usually shows dilatation of the bloodvessels; often it is inflamed and chronically thickened, participating, on the one hand, in causing the ascites, and on the other hand, leading to thickening and distortion of the omentum and the mesentery with consecutive thickening and shortening of the small intestine (a reduction of eight to ten feet in length may occur). Tuberculous infection of the peritoneum is common, occurring in 9 per cent. of the fatal cases (Rolleston), and being more common in cirrhotic males than in other male subjects. It is often responsible for the fatal issue. It is sometimes latent and found only at the necropsy; in other cases it dominates the clinical picture. The pancreas¹ is enlarged, swollen, and fibrotic, but there is no constant relationship between the size and the degree of fibrosis of the pancreas and of the liver; the parenchymatous cells show fatty degeneration and pigmentation; the islands of Langerhans are intact, except in the cirrhosis of hæmochromatosis, in which they become destroyed, occasioning bronzed diabetes.

The kidneys show deviations from the normal in about one-half of the cases. In most of these cases the kidneys are simply enlarged—doubtless the result of overwork (excessive drinking on the part of the patient, and efforts to remove toxic substances circulating in the blood); or they exhibit the lesions of chronic passive congestion (failure of the heart). In some cases, and usually early, albuminuria is encountered; later, interstitial nephritis (arteriosclerotic kidney) occurs in 22.5 per cent. of the cases (87 of 387 cases collected by Rolleston). Few of these cases are due to the cirrhosis per se; both are usually due to a common cause (alcoholism), and both, as well as arteriosclerosis (which is often associated), occur at the same period of life, and predominantly in the male sex. There is no constant relationship between the degree of fibrosis and the size of the kidneys and the liver.

The heart is often fibroid and fatty (alcoholism, arteriosclerosis), and consequently dilated. Endocarditis may occur as an accidental complication and the pericardium may contain a considerable excess of fluid (partly inflammatory, partly transudation). Arteriosclerosis is very common, occurring in more than 60 per cent. of cirrhotic subjects; but it is not due to the cirrhosis per se (the blood pressure is low in cirrhosis); both are more likely due to common causes—alcoholism, male sex, etc.

The lungs exhibit no characteristic lesions; but with the peritoneum they constitute the most frequent seat of tuberculosis in cirrhotic subjects. This tuberculosis may be acute or chronic, latent or obvious; and it leads directly to death in from 12 to 15 per cent. of the cases of cirrhosis (Rolleston). Right-sided pleuritis, due to transport of inflammation through the diaphragm from a perihepatitis, occurs in about 10 per cent. of the cases.

Pathogenesis.—In portal cirrhosis, in consequence of the activity of some poison, larger or smaller areas of the liver tissue, that is, of the cell mantles about the central veins, become necrotic and are removed—whereby the central

¹ Consult Lando, *Zeitschrift für Heilkunde*, 1906, xxvii, 1.

veins become more or less uncovered, thus on cross-section appearing eccentrically situated in more or less misshapen lobules; in some cases in which the destruction of liver cells has been through the entire radius of the cell mantle, the central vein may be entirely exposed and may even be found in the interlobular connective tissue, in which event it is very likely to be mistaken for a branch of the portal vein. The cells destroyed are those about the periphery of the lobules—in the area of portal distribution, conforming to the fact that the etiological factor (poison) is usually carried by the portal vein. Following the primary destruction of certain liver cells, the intact cells, exhibiting a remarkable regenerative capacity, attempt to replace the lost cells and sometimes succeed in restoring more or less completely the volume of the cell mantles. The cells that regenerate are those in the immediate vicinity of those that have become necrotic, that is, in the ordinary case in which the destruction is not widespread, cells toward or at the periphery of the lobule—which thus are not only most exposed to the deleterious influence of poisons carried by the circulation, but in consequence of being better supplied by arterial blood are also better adapted for repair. The writer is inclined to believe, in view of Opie's researches on the circulation, that the primary destructive action may be exerted, at least sometimes, in the so-called midlobular zone—at all events, not immediately at the periphery; that the peripheral cells, being better supplied with arterial blood, survive when others nearer the centre of the lobule succumb, and that these peripheral cells also serve as a focus of regeneration, although, of course, the cells nearer the centre of the lobule also regenerate. One is somewhat confirmed in this opinion by the characteristics and disposition of many of the pseudobiliary canaliculi, which are often most numerous in the skeleton of those lobules that have been much destroyed, and seemingly grow from the periphery into the lobule, in a manner quite analogous to Ponfick's and others' descriptions of their growth into granulation tissue after destruction of the liver cells. As is now generally conceded, these pseudobiliary canaliculi sometimes grow from preëxisting bile ducts, but they also develop from liver cells, which in this respect are histogenetically equivalent to the epithelium of the bile ducts. It is also not unlikely that some of the surviving peripheral cells by proliferation give rise to certain of the hyperplastic islets of liver tissue devoid of central veins.

The continuing activity of the etiological factor leads to a continuous or recrudescent death of certain old as well as newly formed liver cells, which in turn is followed by renewed regeneration. This continuous degeneration and regeneration ultimately lead to complete transformation of the architecture of the liver; certain lobules are entirely destroyed, others are partly or completely rearranged, some are markedly hyperplastic although tolerably well arranged, some show dendritic and other irregular proliferation, new islets of liver tissue entirely devoid of central veins appear, and, what is especially important, new circulatory conditions—capillary and venous—arise.

Concurrent with the degeneration of the liver cells, the framework of the lobules collapses and participates in the formation of the fibrosis. This fibrosis, formed as previously described, ultimately contracts, and on the one hand causes atrophic changes in the liver cells, and on the other obstruction and obliteration of some of the interlobular and intralobular branches of the portal vein; this latter, although it contributes to, is not the sole cause of,

the ascites—other important factors being the aforementioned new circulatory conditions, especially certain noteworthy changes in the hepatic arterial supply. Ultimately the potentiality for regeneration possessed by the liver cells gradually lessens and is finally lost, the general functional capacity of the liver cells being meanwhile compromised by the contracting connective tissue; the degenerative changes predominate over the regenerative, and the patient soon dies, if he has not succumbed earlier to an accidental infection, to which his depressed vitality renders him especially susceptible.

Symptoms.—Cirrhosis of the liver may remain for many years entirely latent; indeed, in most cases the lesions in the liver are well advanced before the onset of noteworthy symptoms, and the disease not infrequently constitutes an unexpected finding at the necropsy table. Symptomatically the disease has been divided into the pre-ascitic and the ascitic stage; and the symptoms are described as being (1) obstructive and (2) toxic. As long as the collateral circulation is efficient obstructive symptoms are in abeyance.

In a few cases, during the early stages of the development of the disease, phenomena attributed to an ill-defined, so-called, active hyperæmia of the liver are encountered; that is, the patient complains of ill-defined or vague gastric symptoms, a sense of weight in the right hypochondrium, perhaps short transitory attacks of slight fever, slight jaundice, and actual pain in the region of the liver, and the liver on examination is found to be enlarged and tender. Such attacks may last several days, disappear, and recur at longer or shorter intervals.

In most cases, however, the early symptoms are an expression of gastro-intestinal catarrh—due partly to portal congestion, and partly, in many cases at least, to chronic alcoholism with acute exacerbations. The patient complains of coated tongue, irregular appetite, flatulence, epigastric distress, especially after eating, sometimes of nausea and vomiting (often in the morning and usually referable to alcoholism), irregular action of the bowels (alternating attacks of diarrhœa and constipation), or habitual constipation, hemorrhoids, etc. The increasing portal obstruction leads to increasing congestion of the gastro-intestinal tract and the spleen, the congestion being doubtless favored by the absence of valves in the portal tributaries. This congestion reduces the functional capacity of the stomach and intestines, limiting the digestive juices and the intestinal secretions (although sometimes increasing the mucus), reducing the absorbing power of the intestines, and often inhibiting peristalsis. Constipation increases and tympanites not infrequently ensues, and both may become of high grade and distressing; occasional relief is sometimes afforded by attacks of diarrhœa which relieve the engorged vessels. These phenomena, while not diagnostic, are highly suggestive of the disease, especially in association with a history of alcoholism.

With the progress of the disease the foregoing evidences of derangement of the gastro-intestinal tract become more marked, and the general health becomes impaired; but the first obtrusive manifestation of the disease, that which induces the patient to seek medical advice, is not infrequently hæmatemesis or ascites.

Hæmatemesis is an initial symptom in some cases, and it is a common event some time in the course of most cases. Usually it occurs early—while the liver is large; when not the initial symptom, it is often the first evidence of serious disease (33 per cent. of the cases); but it may occur late in the disease, that is, after the development of ascites and when the liver may have

become small. The vomiting of blood may come on without warning, especially when the bleeding occurs from œsophageal varices and the blood does not enter the stomach; in some cases it is preceded by distress or pain and faintness, particularly when it comes from the stomach; severe collapse is uncommon; an immediately fatal result is unusual, but it does occur: in one-third of 60 fatal cases collected by Preble¹ the first hemorrhage was fatal. The hemorrhage, as a rule, is large and consists usually of dark, partially clotted blood. A second or a third hemorrhage may occur at short intervals, and perhaps lead directly to death; or repeated hemorrhages may occur for years, eleven years being the maximum (Preble). The hemorrhage may be derived from: (1) Rupture of varicose veins in the œsophagus. These were present in 80 per cent. of the cases collected by Preble; in more than half of these the point of rupture was obvious to the naked eye, but in only 60 per cent. of the cases with œsophageal varices were there the typical signs and symptoms of cirrhosis. (2) Rupture of varicose veins of the stomach, which usually are situated near the cardiac end; the bleeding is sometimes profuse and may lead to a fatal termination. (3) Gastritis, in which event the hemorrhage is due to oozing from distended capillaries and venules. (4) Abrasion of the gastric mucosa, which may proceed to definite ulceration, and is often associated with gastritis. If the hemorrhage does not soon lead to death, it is an important factor in the development of more or less severe anæmia; occasionally, however, it results in at least temporary relief to the patient, relieving the engorged portal vein and its tributaries, leading to reduction in the size of the spleen, and lessening or even preventing the development of ascites.

With the hæmatemesis there is usually blood in the stools; but blood may occur in the stools without the vomiting of blood, especially when the bleeding into the stomach is slow, and also when, as is not uncommon, the source of the blood is the intestine or the rectum or hemorrhoids. In suspected cases of cirrhosis examination of the stools for blood (even occult blood) should be early resorted to.

Hemorrhage from or into other parts of the body is very common. The tendency of cirrhotic subjects to bleed independently of stasis and the presence of jaundice is well known, and is justly attributed to toxæmia with disturbed nutrition, the result of functional insufficiency of the liver. This is doubtless the underlying factor in some of the gastro-intestinal hemorrhages and in much of the other hemorrhages. Epistaxis and bleeding from the gums are very common. Sometimes the amount of blood lost is large; it may occasion a well-marked anæmia, and it has proved fatal. Not infrequently the blood is carried to the fauces or pharynx, and, being then spat out, may simulate hæmoptysis. The larynx occasionally exhibits varices (usually associated with œsophageal varices) which rupturing may lead to severe hemorrhage or a fatal issue. Bleeding from the lungs is not uncommon. Menorrhagia and metrorrhagia occur particularly in the early stages. Hemorrhages, usually petechial, into or beneath the skin, the mucous membranes (nasal, pulmonary, urinary, etc.), and the serous membranes (pleura, pericardium, peritoneum) are not rare, although not as common as in biliary cirrhosis.

Jaundice is not a part of portal cirrhosis, but it occurs as a complication some time during the course of from 15 to 20 per cent. of the cases. A faint

¹ *American Journal of the Medical Sciences*, 1900, cxix, 263.

subicteric hue to the conjunctiva and the skin is not uncommon; it may come and go repeatedly, and last a longer or shorter time; it is usually due to a radicular cholangitis, and has been attentively studied by Naunyn.¹ More marked grades of jaundice attributable to catarrhal inflammation of the biliary ducts are occasionally observed. In the terminal stages more or less marked jaundice may result from associated catarrh of the biliary ducts, duodenal catarrh, compression or kinking of the biliary ducts or degenerative changes in the liver cells and the associated changes of acute yellow atrophy.

Toxic symptoms may develop at any time during the course of the disease. They are much less common early than late, but they are occasionally observed early in cases that for some time pursue a latent course. Minor symptoms consist of restlessness, weakness, headache, and itching; the itching may precede or be unassociated with jaundice. More severe toxic symptoms comprise: (1) Apathy, stupor, and coma; (2) an active noisy delirium; and (3) convulsions, paralyses, contractures, one of which may alternate with the others. These phenomena much resemble and are often mistaken for those due to uræmia; in fact, they are not infrequently related to renal insufficiency. The nature of the poison is not known, the condition being ascribed merely to hepatic insufficiency (hepatargia); but since the symptoms not infrequently develop in the absence of jaundice, they cannot be attributed to cholæmia in a narrow sense.

Cirrhosis of the liver may be afebrile throughout its course; during the terminal stage the temperature may be subnormal; but attacks of fever (100° to 102.5°) are not uncommon. Sometimes these seem to be part of the disease, especially when it advances rapidly; sometimes they are due to attacks of perihepatitis; sometimes they are due to complications. The more continuous fever, which is rather common late in the disease, is usually due to tuberculosis—of the peritoneum, lungs, etc.

Physical Signs.—The general aspect is often quite characteristic and may suggest the diagnosis. In the early stages there is pallor and sallowness; the face is peculiarly muddy or subicteroid. Later, the patient becomes thin and emaciated; the musculature is soft and flabby; the general integument is dry, harsh, and wrinkled; the face is drawn, occasionally bloated; the eyes are sunken; the conjunctivæ are muddy or subicteric; the cheeks and temples are hollow; the lips are dry and fissured; the tongue is flabby and coated; the gums are spongy and bleed readily; the breath is foul; the skin of the face and cheeks reveals many distended venules that are prone to bleed; nævi consisting of localized stellate varices and so-called mat nævi ("areas of skin of a reddish or purplish color due to the uniform distention of small venules," Osler) may occur on the face, neck, and back; and small capillary, subcutaneous hemorrhages are prone to develop, especially in regions exposed to traumatism. The abdomen is more or less markedly, sometimes enormously, distended, and is in notable contrast to the emaciated face and extremities. The umbilicus becomes everted and has been known to rupture. The abdominal wall reveals enlarged and varicose veins due to the establishment of a collateral circulation. The very obvious communication sometimes established between the superficial epigastric veins and the long thoracic and the mammary veins is due not to the portal obstruction, but to interference

¹ *Internationale Beiträge zur inneren Medizin*, von Leyden, 1902, i, 457.

with the circulation in the inferior vena cava the consequence of increased intra-abdominal pressure caused, as a rule, by the ascites. The collateral circulation, although often marked, is rarely ample or efficient; sometimes apparently it is for a time, at least, since an extensive collateral circulation occasionally relieves more or less permanently a previous ascites, but under these circumstances one cannot ignore the likelihood of the liver having become more pervious to the passage of the portal blood.

Examination of the liver shows it to be enlarged in most cases, but the size varies considerably in different cases and in the same case at different times. Foxwell has well said that the liver is "enlarged at all stages of the disease, and that whether enlarged or contracted the clinical symptoms and course are much the same," a statement that has been especially emphasized by Dreschfeld, Rolleston, etc., and is in conformity with my own experience. Usually before the onset of the ascites, and often during the prevalence of the ascites, the lower border of the liver is palpable one, two, or three fingers' breadth below the margin of the ribs in the right nipple line. The liver is often tender, its edge sharp, and its surface irregular; in some emaciated subjects the hobnail surface can be readily appreciated by the palpating hand, but one must be careful not to confuse therewith irregular areas of fat and fibrous tissue in the abdominal wall. In some cases late in the course of the disease percussion and palpation show the liver to be considerably reduced in size, but in many cases in which the liver appears to be small it is of normal or increased size and elevated, being pushed up by ascites or drawn up by a parietic diaphragm (due to perihepatitis, etc.); in other cases emphysema may decrease the extent of liver dullness, or adhesions may prevent the descent of the organ on deep inspiration. In any event, whether small or only elevated, the organ can often but not always be palpated by firm pressure upward under the ribs. The left lobe is often disproportionately small and not at all palpable in the epigastrium—which is quite significant of cirrhosis. Notable variations in the size of the liver occur from time to time, and often rather rapidly; the common change consists of the reduction in size of a large liver, but the reverse is not unknown, although it may be temporary.

The spleen is usually palpable two or three fingers' breadth below the left costal margin; but as also in the case of the liver, tympanites and ascites often interfere with satisfactory examination. Early the spleen is sometimes not notably increased in consistency; later it is usually firm, and it is often tender. It is often much reduced in size by copious gastro-intestinal hemorrhage, severe diarrhoea, the recurrence of ascites after tapping, etc. On the contrary, an unusual enlargement is sometimes a forerunner of hemorrhage, which may be prevented by a prompt resort to free purgation.

Ascites is often the first obtrusive manifestation that leads the patient to seek medical advice. Sometimes it is attributed to a definite exciting cause, such as exposure, cold, trauma, etc.; but, as a rule, the onset is gradual and without definite cause, and the accumulation is slow. Occasionally the fluid accumulates with marked rapidity, in which event it is attributable to thrombosis of the portal vein. Ascites is to be looked upon as a late event, although it is not improbable that congestion and transudation of a small amount of fluid into the peritoneum occurs comparatively early, in some cases at least; but this is more or less readily absorbed by the lymphatics of the diaphragm and the parietal peritoneum. When, however, these are overtaxed and the collateral circulation proves insufficient, ascites ensues. It is present in

almost if not quite all subjects dying of cirrhosis per se, but in scarcely more than 50 per cent. of cirrhotic subjects at the necropsy table. Klopstock found ascites in 172 of 250 cases of cirrhosis that came to necropsy. There is no direct relationship between the size of the liver and the onset or the degree of ascites; in most cases the liver is larger than normally at the time of development of the ascites; but a large liver may be present without ascites; a small liver is rarely found without ascites. It is believed by many observers, such as Hale White, Rolleston, etc., and the writer is inclined to concur in the opinion, that ascites is really a terminal event, and that if, in a case presumed to be cirrhosis of the liver, the patient lives to require a second tapping, the cirrhosis is complicated with chronic peritonitis (perihepatitis), or the diagnosis is incorrect.

Some patients require repeated tapplings. Casati reports one in whom tapping was done 111 times, and Rumpf a case in which it was done 301 times; but these are most unusual. Usually the tapping is of temporary benefit, relieving the patient's distress for a short time only; the fluid soon re-accumulates, slowly, as a rule, occasionally rapidly. In unusual cases more or less permanent benefit seems to result, doubtless the consequence of the formation of adhesions. Cheadle reports a case in which a man was tapped 19 times and lived eight years after the last tapping; and MacDonald reports two cases in which disappearance of the ascites ensued upon 31 and 60 tapplings, respectively.

The ascitic fluid varies in amount—in the average case from 4 to 8 or 10 liters, but it may be as much as 20 liters, with consequent enormous distention. The fluid is usually pale amber in color, clear, somewhat opalescent, and alkaline; it has a specific gravity of 1008 to 1015, and it contains 0.5 to 1 per cent. of albumin, and occasionally also traces of urea, urobilin, purin bodies, sugar, etc. Sometimes it is turbid from associated peritonitis, in which event it is of higher specific gravity and contains more albumin; rarely it is chylous, chyloform, or hemorrhagic—the last mentioned usually the consequence of traumatism, or petechial hemorrhages in the serosa, etc. Bacteriological examination of the fluid shows it to be sterile, unless there has been some secondary infection. Cytological studies show the cellular constituents to be few and to consist of lymphocytes and endothelial cells, with a few erythrocytes and leukocytes. Cryoscopy reveals the freezing point to vary with the amount of albumin.

Varying with the amount of fluid there is more or less displacement and compression of the abdominal and thoracic organs. The liver and the diaphragm are pushed upward and cause embarrassment of the respiration and the heart action, and not infrequently compression and consequent congestion and collapse, and perhaps bronchopneumonia, of the bases of lungs, particularly the right.

The ascites is doubtless due to different factors: portal obstruction, peritonitis (or perihepatitis), and toxæmia—which are variously operative in different cases. Portal obstruction is an important, but by no means the sole, factor. Ascites seems a natural consequence of the other manifestations of portal obstruction (hæmatemesis, distended veins, collateral circulation, etc.), and it is the conspicuous result of complete portal obstruction, such as occurs in portal thrombosis. The cause of the increased portal pressure in cirrhosis is not immediately patent, and probably it is not always the same. In some cases, as previously mentioned, thrombosis of at least some of the

portal radicles is found upon histological examination of the cirrhotic liver—which serves to explain, in part at least, the increased portal pressure, in some but probably few cases. Usually the increased portal pressure is attributed to obstruction of the portal vessels by the newly formed connective tissue, but doubt has been thrown upon this by Kretz and more recently by Herrick,¹ who believes that an important factor in this increased portal pressure is the communication of the arterial pressure to the portal circulation. Herrick found experimentally that a portal cirrhotic liver gives passage to an amount of portal fluid proportionate to its weight, and that variations in the pressure in the hepatic artery from 0 to 130 mm. Hg. causes, in the normal liver, a rise of only 3 or 4 mm. Hg. in the portal pressure, but in the cirrhotic liver, a rise of from 20 to 30 mm. Hg. in the portal pressure. This is due to the fact that the arterial supply to the fibrous tissue of the cirrhotic liver is increased, whence an increased amount of blood flows through the arterial capillaries, and to the fact that there is a far freer communication between the arterial and the portal currents in the cirrhotic than in the normal liver, in consequence of which the arterial pressure through the medium of dilated capillaries is communicated to the portal vessels. Lessening of the portal area, apparent upon histological examination of the liver, is probably also of some significance in increasing the portal pressure. That increased portal pressure is not the sole factor in causing ascites is apparent from the fact that it is not always present when the portal pressure is undoubtedly high, as at the time of (or just before) severe gastro-intestinal hemorrhages; in these cases, however, the hemorrhage is often due to factors other than the increased portal pressure, such as inflammation and erosion of vessels (under pressure); and frequently the peritoneal lymphatics are still active and able to carry away a considerable amount of fluid. Usually the fluid is not inflammatory in nature, but sometimes it is, supporting the view that the ascites is sometimes due in part at least to peritonitis (perihepatitis). In part the ascites may be toxic, an unknown toxin acting as a lymphagogue on the peritoneum; but this is pure hypothesis.

Œdema is common late in the disease, that is, it comes on after the ascites. It is most marked in the feet and legs, but often involves the trunk, especially the dependent portions; rarely anasarca ensues. The œdema of the lower extremities is referable to interference with the venous flow in the inferior vena cava. Œdema of the anterior abdominal wall is sometimes related etiologically to increased pressure in the dilated and tortuous regional veins. Occasionally œdema precedes the development of the ascites, in which event it may be due to one of several factors: it may be toxic, in nature analogous to some of the ascites (hypothesis); it is sometimes due to anæmia or to chronic renal or heart disease with dilatation; occasionally it may result from thrombosis of the iliac veins or the inferior vena cava; or it may be due to alcoholic or other toxic neuritis. Anyone or several of these may be a factor in causing the later œdema, that is, that developing after the ascites.

The action of the heart is often impeded by the upward pressure exerted by the ascites. The blood pressure is usually low, the pulse small and frequently rapid, and the breathing labored. The myocardium is often weak, due to fibroid alterations the consequence of alcoholism, malnutrition, and cachexia. A systolic murmur and other evidences of dilatation may develop.

¹ *Journal of Experimental Medicine*, 1907, ix, 93.

Occasionally there is a functional murmur in the pulmonary area, due to anæmia or to kinking of the pulmonary artery the consequence of displacement of the heart; often it disappears after removal of the ascites and the return of the heart to its normal position. A venous murmur is sometimes audible in the epigastrium (dilated veins in the falciform ligament), and sometimes over the spleen. Evidences of cardiac failure are sometimes so marked as to obscure the cirrhosis.

The blood shows the changes of secondary anæmia. There is no leukocytosis unless there is some associated infection, which is not rare. Late in the disease evidences of considerable toxic disorganization of the blood become apparent—hydræmia, cedema, submucous and subcutaneous hemorrhages, etc., and the blood findings rarely may simulate those of pernicious anæmia.

The urine is usually diminished in amount, in consequence of deficient absorption of fluid, the large amount of fluid in the peritoneum, pressure exerted on the kidneys, and decreased blood pressure; the specific gravity and the color are high, due to concentration; sometimes the urine is reddish in color (urobilin, urates); it is highly acid; sometimes it contains albumin (congestion due to failing heart, parenchymatous changes, nephritis); sugar is rarely present; the chlorides are diminished; the urea is reduced; the ammonia is increased (effort to neutralize acid intoxication); indican, albumoses, leucin, tyrosin, and increased volatile fatty acids may be found. Alimentary levulosuria may be detected, and may prove a serviceable sign of hepatic insufficiency; which is true also of urobilinuria and phenoluria. In so-called bronzed diabetes (hæmochromatosis) sugar is present in the urine, but it is due not to the associated cirrhosis of the liver, but to fibrotic and other changes in the pancreas.

Complications.—The cirrhotic subject is peculiarly susceptible to infections of diverse sorts, both acute and chronic. Tuberculosis is the most common (more common in adults than in children) and shows a special predilection for the lungs and the peritoneum. The complication is often overlooked, because unthought of and unsought; it should always be suspected when there is fever otherwise unaccounted for. In some cases the lung involvement is altogether latent; in other cases it gives rise to physical signs sufficient for diagnosis, but no obtrusive symptoms; in a few cases it dominates the clinical picture. Tuberculous peritonitis is very common, and not infrequently determines the fatal issue. It is often overlooked, the ascites naturally being attributed to the cirrhosis *per se*; it is suggested by the presence of fever, tenderness and rigidity of the abdominal wall, perhaps increase in the amount of ascites previously stationary (the fluid sometimes becomes hemorrhagic), more rapid course, and more speedy death. The ascites seems to predispose to tuberculous infection, which otherwise is uncommon in adult males. The cirrhosis is undoubtedly the primary disorder in the great majority of cases, but some observers believe that in some cases the tuberculosis of the peritoneum is the primary, the cirrhosis the secondary.

Tuberculous pleuritis is not rare, but non-tuberculous pleuritis also occurs. The pleuritis may be primary or secondary to disease of the lungs or the peritoneum; in the latter case the infection doubtless travels through the diaphragm. The pleuritis is usually right-sided, but it may be left-sided or bilateral. Some care is required in diagnosing right-sided pleuritis, since compression of the lung by a large liver or ascites may occasion dulness

and other signs simulating pleural effusion; or a localized bronchitis or bronchopneumonia may develop.

Evidences of involvement of the kidneys occur in about one-half of the cases. In some cases there is only a slight albuminuria, attributable to toxic degeneration of the renal epithelium or passive congestion; in other cases in addition to the albuminuria, which is more marked, there are signs significant of parenchymatous nephritis; in most cases (almost one-fourth of all cases of cirrhosis) the common manifestations of interstitial nephritis occur. This nephritis is scarcely due to the cirrhosis as such; it is rather the result of alcoholism and other causes that prevail at the time of life that cirrhosis is most common.

Peripheral alcoholic neuritis (neuromuscular pains and tenderness, muscular cramps, cutaneous hyperæsthesia, lost knee-jerks, etc.) occurs in a considerable proportion of cases, and is often overlooked or the symptoms are misinterpreted. A number of acute infections, especially pyococcic infections (local and general), endocarditis, croupous pneumonia, etc., are not uncommon, and may determine the fatal issue.

Diagnosis.—The diagnosis of cirrhosis of the liver in the early stages is not always an easy matter, but it should be made more frequently than it is. In an alcoholic subject with dyspepsia, an enlarged and tender liver suggests the diagnosis; and this is virtually confirmed if to these there be added an enlarged spleen, perhaps recurring attacks of pain in the region of the liver, with slight fever or slight jaundice, and urobilinuria. If the diagnosis were made upon these symptoms and the patient treated accordingly, many subjects doubtless would not later exhibit the typical phenomena of advanced cirrhosis; in some perhaps the diagnosis would be incorrect, but in many the disease would be arrested—of the possibility of which the lesions in the hepatic parenchyma afford abundant testimony. Later, when hæmatemesis, ascites, the hepatic facies, etc., have developed, the diagnosis can be no longer in doubt.

Little difficulty is likely to be experienced in differentiating biliary cirrhosis, which, aside from its rarity, occurs in younger, usually non-alcoholic subjects, and is characterized by persistent jaundice, an enlarged smooth liver, much enlarged spleen, the absence of all signs of obstruction of the portal circulation, and a very chronic course.

When the liver is enlarged and there is no ascites, one must exclude other causes of enlargement of the liver, such as passive congestion (chronic cardiac or pulmonary disease, cyanosis, usually marked enlargement and tenderness of the liver, some jaundice, urinary signs of congestion of the kidneys, etc.); fatty liver (rather soft and smooth liver, causes of fatty disease of the liver, such as tuberculosis, etc.); amyloid disease (causes of amyloid disease, involvement of other organs, spleen, kidneys, intestines, etc.); leukæmia (examination of the blood, etc.); malaria (history of chronic malaria or repeated acute attacks, plasmodia or pigment in the blood, disproportionate enlargement of the spleen, irregular fever, cachexia, anæmia, response to quinine, and perhaps absence of a history of alcoholism). Syphilis is suggested by a history or other evidences of syphilitic infection, more local distress and pain due to the more marked perihepatitis, irregular enlargement of the liver due to a gumma or irregular depressions due to stellate scars, and more common jaundice due to compression of the biliary ducts; in some cases, however, the differential diagnosis is quite

impossible, in which event recourse should be had to antisyphilitic treatment.

The occurrence of hæmatemesis and other gastro-intestinal hemorrhages necessitates differentiation from gastric and duodenal ulcer. This is often difficult, but cirrhosis is suggested by a history of alcoholism, enlarged and tender liver, an enlarged spleen, absence of marked evidence of hyperchlorhydria (hyperchlorhydria may occur in cirrhosis), absence of localized epigastric tenderness, etc. In some cases of cirrhosis the hemorrhage is due to associated gastric or intestinal ulceration, which, however, is commonly not of the peptic variety. Hæmatemesis, especially if small in amount, may also suggest carcinoma of the stomach; but in cirrhosis there are no signs of pyloric obstruction or the evidence of carcinoma afforded by the gastric contents and a tumor is not palpable; indeed, in cirrhosis there is more likely a void in the epigastrium rather than a sense of increased resistance. In the event of gastric carcinoma signs of involvement of the liver are likely to supervene sooner or later, so that ultimately the differential diagnosis has to be made between cirrhosis and secondary carcinoma of the liver; alcoholism, long-continued dyspepsia, and enlargement of the spleen are much in favor of cirrhosis. Should the primary growth be elsewhere than in the stomach, there may be localizing signs or symptoms, and perhaps metastases elsewhere, as in supraclavicular or other glands, etc. Splenic anæmia, or Banti's disease, may be excluded by the absence of recurring hemorrhages with intervals of comparative health, of primary enlargement of the spleen, of early anæmia, and of later development of signs of portal obstruction, and the comparative youth of the subjects—twentieth to the fortieth year.

When ascites has developed one must exclude other causes of ascites, especially chronic peritonitis and perihepatitis (multiple serositis), thrombosis of the portal vein, tumors of the peritoneum and the abdominal organs, chronic cardiac disease, and cachectic states. As already stated, the ascites in many cases of cirrhosis is due, in part at least, to peritonitis or perihepatitis, but in other cases ascites may be due solely to perihepatitis, occurring alone or as part of multiple serositis, or in tuberculous peritonitis. Tuberculous peritonitis is suggested by the absence of a history of alcoholism and of evidences of portal obstruction, and by the presence of fever, tenderness and rigidity of the abdominal muscles; should the tuberculous peritonitis be secondary to cirrhosis with ascites, there may be a rather sudden increase in a previously stationary ascites, a more rapid course, and speedy death.

Thrombosis of the portal vein occurs in one-third of the cases of cirrhosis, and is suggested by sudden development of ascites (or sudden increase in the ascites, if already present), rapid enlargement of the spleen, severe (perhaps repeated) hæmatemesis, and rapid recurrence of the ascites after removal; the sudden onset of these symptoms in the absence of a history of alcoholism suggests primary thrombosis of the portal vein, but in many cases of supposed primary thrombosis there has been a latent cirrhosis until this event.

In the ascites of tumors of the peritoneum or the abdominal viscera, the seat of the primary growth may be more or less obvious, metastases may be found in the supraclavicular fossa, in glands, about the umbilicus, along the course of the falciform ligament of the liver, etc.; emaciation and cachexia are more marked than in cirrhosis, and signs of portal obstruction are wanting; should the liver or biliary ducts be involved, there is also deep jaundice and nodular and progressive enlargement of the liver.

The ascites of chronic cardiac disease should be recognized by detecting the disorder of the heart or an antecedent pulmonary disorder with consecutive hypertrophy and dilatation of the heart; the œdema of heart disease begins usually in the legs and other dependent portions of the body (only exceptionally giving rise to primary ascites); and the œdema and ascites, as well as the size of the liver, vary with the functional activity of the heart, responding, therefore, to cardiac tonics.

The ascites of cachectic states, such as advanced nephritis, amyloid disease, leukaemia, various anæmic conditions, convalescence from typhoid and other infections, is usually slight in grade, often only part of general anasarca, and is associated with readily recognizable disorders; only when there is associated cardiac or hepatic disease does the ascites become at all marked.

In some cases of ascites the liver is small—in which event the disorder may be syphilis of the liver, advanced passive congestion (red atrophy), chronic deforming peritonitis and perihepatitis, thrombosis of the portal vein, etc. Each of these should be readily differentiated by attention to the aforementioned signs and symptoms. The small size of the liver may also be due to acute yellow atrophy superadded to cirrhosis of the liver—which also should be readily recognized.

Examination of the fluid removed by tapping (or otherwise) also aids in the differential diagnosis. The transudate of cachectic states is clear, pale amber colored, and limpid; it has a specific gravity of 1010 or less, and contains less than 1 per cent. of albumin; its cellular content is slight and consists of a few lymphocytes and endothelial cells, and perhaps an occasional erythrocyte. The transudate due to mechanical obstruction is clear, amber colored; it has a specific gravity of 1008 to 1014, and contains from 1 to 3 per cent. of albumin; its cellular content consists of a few lymphocytes and endothelial cells, a few erythrocytes, and an occasional polynuclear leukocyte. The inflammatory exudates may be serous, serofibrinous, purulent, or hemorrhagic in character; even the serous exudates are usually slightly turbid; they have a specific gravity of 1015 or more, and they contain from 3 to 6 per cent. or more of albumin; their cellular content consists of lymphocytes, polynuclear leukocytes, endothelial cells, and erythrocytes in varying proportion, depending upon the nature of the infection; lymphocytes in large numbers (upward of 90 per cent.) indicating tuberculous infection, disproportionate polynuclear leukocytosis indicating pyococcic infection. In tuberculous peritonitis and in malignant disease of the peritoneum the fluid is often quite obviously hemorrhagic. The tuberculous nature of the process may be further demonstrated by culture methods, animal inoculations and resort to tuberculin reactions. That the fluid is due to malignant disease may be proved by finding cells with asymmetrical karyokinetic figures. Occasionally the fluid is chylous (admixture with chyle) or chyliform (fatty degeneration of the endothelial and other cells).

The aforementioned figures relating to the specific gravity and the percentage of albumin of the ascitic fluid are often modified by the occurrence in the one patient of two or more conditions that give rise to ascites. The percentage of albumin in the fluid may be calculated from the specific gravity by means of the formula of Ruess, which is said to be subject to an error of less than 0.25 per cent.: the percentage of albumin equals $\frac{2}{3}$ of the specific gravity minus 1000 (that is, $\frac{2}{3}$ of the last two figures of the specific gravity) less 2.8.

Special Types of Cirrhosis.—Certain special types of portal cirrhosis may be differentiated, of which the following are the more important: (1) Cirrhosis of hæmochromatosis; (2) malarial cirrhosis; (3) anthracotic cirrhosis; and (4) cirrhosis in children.

Cirrhosis of Hæmochromatosis (or Bronzed Diabetes).—In 1871 Troisier directed attention to a diabète bronzé, which was later studied by Hanot and Chauffard, Letulle, and others of the French school, who spoke of it also as cirrhose pigmentaire diabétique. In 1899 von Recklinghausen, under the designation hæmochromatosis, described a general pigmentation of the viscera—which was soon recognized as identical with the bronzed diabetes of the French. It is a rare disease: Futeher,¹ in 1907, was able to find references to only 35 undoubted cases; several additional but more or less doubtful cases (in which diabetes did not develop) have also been reported.

The disorder is most common in men (33 of 35 cases), and occurs between the ages of thirty and sixty-one years, but it is most common during the fifth decade. Its characteristics are pigmentation of the viscera and usually also of the skin, cirrhosis of the liver, and fibrosis of the pancreas with involvement of the islands of Langerhans and consequent diabetes. The pigmentation is generally held to be derived from hæmolysis of the erythrocytes, but the nature of the hæmolytic agent is not known: it is perhaps auto-toxic (possibly intestinal in origin) or bacterial; it is believed not only to cause hæmolysis, but also to produce degenerative and other changes in the cells of the liver, pancreas, skin, etc., and to influence their metabolic activities so that they convert the soluble hæmoglobin into insoluble hæmosiderin and hæmofuscin. A contrary and less held opinion is that the pigmentation results from some metabolic defect whereby iron is not eliminated from the system, an opinion that finds some support in the absence of general hæmochromatosis in pernicious anæmia in which there is hæmolysis. These pigments become deposited in the tissues, usually together; but the iron-free pigment may predominate, as in the skin, for instance. The visceral pigmentation antedates that of the skin. The liver, as a rule, is most markedly affected. In the liver and the pancreas, especially, fibrotic changes develop with the pigmentation and degenerative changes in the parenchyma; in advanced cases these reach a high grade, and in the pancreas ultimately involve the islands of Langerhans and lead to diabetes (but death may ensue before this happens). The sequence of events, therefore, may be described as hæmolysis, chronic interstitial hepatitis and pancreatitis, and diabetes. The diabetes is undoubtedly a late event and not the primary condition as originally maintained by the French. Macroscopically the liver is usually enlarged and presents the common appearances of portal cirrhosis; it is, however, manifestly pigmented, reddish, resembling brick dust or iron rust. The spleen also is enlarged, firm, and pigmented.

The *symptoms* are those of cirrhosis in its early stage, especially the dyspeptic symptoms and pain in the right hypochondrium; usually also there is progressive weakness. Occasionally the onset may be sudden. The noteworthy signs consist of general cutaneous pigmentation (suggesting Addison's disease), enlargement of the liver and the spleen (quite like that of ordinary portal cirrhosis), and the ultimate development of diabetes. Ascites may be slight or absent; hemorrhages may or may not occur, but death may

¹ *American Journal of the Medical Sciences*, 1907, cxxxiii, 78.

result from rupture of œsophageal varices, or there may be purpuric attacks. Diabetes when it supervenes is severe and dominates the clinical phenomena; acidosis is common and usually leads to coma.

The *diagnosis* is apparent from the cutaneous pigmentation, enlargement of the liver and the spleen, and diabetes. The cutaneous pigmentation must be differentiated from that of argyria; of chronic jaundice (in which the scleræ are involved); of Addison's disease (in which there is no enlargement of the liver and spleen and no diabetes); and of oehronosis (alkaptonuria).

The *prognosis* is bad, although life may be prolonged for many years. Death may occur suddenly from rupture of œsophageal varices, etc.; usually it occurs within a year after the appearance of glycosuria.

The *treatment* in the early stages is that of cirrhosis; in the later stages, that of diabetes.

Malarial Cirrhosis.—The lesions that commonly occur in the liver in malaria have been described under acute hepatitis, and attention has been directed to the rarity of malaria as a cause of cirrhosis; indeed, many competent observers doubt that malaria ever causes cirrhosis, preferring to believe that the cases of so-called malarial cirrhosis are due to the operation of the ordinary causes of cirrhosis (alcohol, etc.) in a malarial subject. Malarial cirrhosis unquestionably is rare in temperate climates; but in view of the very marked lesions produced in the liver by acute and chronic malaria, especially severe attacks, there can be no well-founded reasons for doubting that malaria may cause cirrhosis.

Recently, Tucker,¹ studying the subject in India, states: It is not infrequent even in young children, from whose histories one can easily eliminate all the possible contributory factors of cirrhosis, with the exception of malaria, a disease which produces very decided effects on other important viscera. In such patients one invariably finds that the history of ill health and progressive enlargement of the spleen has extended over several years. There have been repeated attacks of remittent fever with intermissions of fair health. After two or three years of this condition the patient notices the enlargement of the spleen. By the time that this organ has reached the level of the umbilicus or has filled up the left side of the abdomen he is suffering from all the symptoms of a profound secondary anæmia. Later still there is profound emaciation, and the abdomen still further distends from the collection of fluid in the peritoneal cavity. This fluid, as a rule, is not very abundant. On examination one can easily feel the anterior edge of the large, stone-like spleen, and can also generally palpate below the right costal margin the lower edge of the liver, which is smooth, hard, and painless. The general condition of the patients varies considerably. In many the fluid does not recur after tapping, or only very slowly, and after several weeks of treatment the patient leaves the hospital considerably improved and with a somewhat smaller spleen. Finally, the patients return with symptoms of profound debility and emaciation, exhibiting the late toxæmic condition seen in the other forms of cirrhosis, and after a few weeks or months they fall into a condition of coma and die.

On opening the abdomen the most prominent object is the enormous spleen. The splenic capsule shows the marks of the repeated attacks of inflammation, of which it has been the seat, in a general thickening, with

¹ *Lancet*, 1908, i, 1474. Consult also Duprey, *Lancet*, 1908, ii, 308.

here and there patches of the color of mother of pearl two or three inches square and perhaps a quarter of an inch thick. In such spots the capsule is often found adherent to adjacent organs, and frequently the peritoneal adhesions have been produced into long bands. The attachment of the spleen to the diaphragm is by broad adhesions or by numerous thread-like bands, or by both; and all such adhesions are very vascular during life. The liver itself is somewhat small, but the shrinking is not so extreme as in the alcoholic cirrhosis. The surface is finely granular, not hobnailed, and the granulations are often not uniform over the whole surface, but here and there smooth areas are to be observed. Remains of the repeated attacks of hepatitis and capsulitis are invariably found in the adhesions of the liver to the diaphragm and sometimes thick peritoneal bands about the transverse fissure. The blood is thin and sometimes only just stains linen; the red blood corpuscles show the ordinary characters of profound secondary anæmia and are about 1,500,000 or 2,000,000. Malarial parasites are never found in the peripheral blood, nor are they found on splenic puncture during life (Tucker).

Anthracotic cirrhosis is a rare disorder of the liver, described by Welch, and associated with the presence of carbon dust (analogous to coalminer's lung). Adami has described a similar condition.

Cirrhosis in children is by no means rare.¹ Usually it is of the common or portal type; it may be of the biliary type; in congenital syphilis there is a so-called pericellular cirrhosis; and fibrotic lesions sometimes designated cirrhosis are found associated with congenital obliteration of the biliary ducts.

The portal cirrhosis of children is due to the usual causes, such as alcohol, intestinal intoxication, infections, etc. It is not uncommon in several members of the same family, which doubtless is referable to familial habits as regards alcohol, etc., and to the influence of hereditary syphilis (pericellular cirrhosis of the liver), which although cured may leave the liver quite susceptible to the common causes of portal cirrhosis; the disorder, therefore, in many cases is undoubtedly parasymphilitic.

The *symptoms* in general conform to those in the adult. Hæmatemesis is less common (greater distensibility of the spleen in children), diarrhœa and slight jaundice are more common; the liver and the spleen are disproportionately larger than in adults (relative greater volume and more ability for repair in children); tuberculous peritonitis is common and perhaps accounts for most of the ascites and the not uncommon fever. In some cases, described by Omerod and Homen, the symptoms are almost wholly nervous (probably syphilitic with lesions of the nervous system).

Prognosis.—On the whole, the outlook in portal cirrhosis is not encouraging—which fact is due largely to the nature of the disease and the advance it has made when usually recognized. There is much evidence, however, that the liver cells early in the disease and often for a long time undergo considerable hyperplasia in an effort to compensate for the damage done, and the collateral circulation is additional evidence of efforts in the same direction; there is reason to believe, therefore, that if early recognized and properly treated, the disease could often be brought to a standstill, perhaps cured. As a matter of fact, when recognized early marked benefit is often obtained.

¹ Consult Jones, *British Journal of Children's Diseases*, 1907, iv, 1, 43; and Pexa, *Wiener klinische Rundschau*, 1908, xxii, 33, 50, 68, 83, 117.

The size of the liver is of comparatively little prognostic significance, although a very small liver is indicative of the later stages of the disease; as already stated, however, the liver may be enlarged from the beginning to the end. The occurrence of hæmatemesis does not make the outlook hopeless, since it is sometimes an early symptom and may lead the patient to mend his ways, in consequence of which he may have no additional symptoms, perhaps for years. With the onset of ascites the prognosis becomes distinctly bad, since ascites must be looked upon as a terminal event in the ordinary course of the disease; sometimes, however, the ascites becomes stationary or subsides, in consequence of the establishment of an efficient collateral circulation, and the patient may live for many years. The prognosis is also bad in the event of anasarca, emaciation, cholæmia, failure of the heart or the kidneys, and any of the many complications and secondary infections. Ordinarily the disease runs a fatal course within three years from the onset of symptoms; but sometimes arrest ensues and the patient may live for a number of years—eight or ten. Occasionally the course is rapid, six or eight months, especially in young subjects much addicted to alcohol; in these cases to the cirrhosis is often superadded serious degenerative changes in the hepatic parenchyma, and a form of severe jaundice may ensue and lead to the fatal issue.

Treatment.—The treatment of portal cirrhosis to be effective must be instituted during the formative stage of the disorder; little beneficial effect can be expected after the destruction of much hepatic parenchyma and the overgrowth of much fibrous tissue. The only trustworthy treatment is based upon our conception of the etiological factors, and consists essentially in the avoidance of all causes known or suspected to lead to the disease. Of these, the most important are alcohol, stimulating and highly seasoned foods, foods likely to undergo fermentation in the intestinal tract, etc., all of which must be strictly prohibited. In the early stages of the disease, when the hope of marked benefit, if not cure, may be reasonably entertained, alcohol in all forms (including medicinal agents, tinctures, etc.) must be absolutely interdicted; there can be no question that the likelihood of improvement or arrest of the disease depends more upon the non-use of alcohol than upon any or all other factors. But when the disease is well advanced and cure or notable improvement is manifestly impossible, it is not always necessary, or wise, to discontinue alcohol entirely: the question of its use or non-use must be decided for each individual patient. Although it may be quite obvious that the patient's deplorable condition is the consequence of the misuse of alcohol, it does not follow as a corollary that the disuse of alcohol will result in improvement; he may have become so accustomed to the effects of alcohol and so dependent upon it for all more or less sustained bodily and mental effort that its withdrawal will be attended by more ill consequences to the general economy than by good results to the liver. In these cases the alcohol should be reduced to the smallest amount consistent with general well-being; and this amount should be given well diluted with the meals. *Nux vomica* or *strychnine* is often an excellent substitute for the alcohol, and is otherwise beneficial; in some cases, after its use for a time, the alcohol may be entirely withdrawn without ill effects to the patient.

The *diet* is of the utmost importance: on the one hand, it must be sufficiently nourishing; on the other hand, it must be absolutely non-irritating to and not overtax the functional capabilities of the liver, and it must be readily

assimilable and not likely to undergo fermentation in the intestine, which, if it occurs, may give rise to the production of autotoxins harmful to the liver. Milk is unquestionably the best diet; it is sufficiently nourishing, readily digested and assimilated, its proteid is easier utilized by the liver than that of meat; it leaves little residue, it usually does not lead to intestinal fermentation and autotoxæmia, and it is somewhat diuretic. In almost all cases it should form the only diet, for a time at least—four to six weeks, depending upon the improvement shown by the patient. Two to three quarts should be given during the twenty-four hours. Sodium bicarbonate or some one of the natural alkaline waters, such as Vichy, Apollinaris, etc., may be added to the milk with advantage; or it may be flavored with vanilla, tea, coffee, chocolate, cocoa, etc. If the milk should seem to disagree with the patient, skimmed milk (in which the fat is reduced from 4 to 1 per cent.) or buttermilk, or koumyss (less desirable on account of the contained alcohol) may be tried; in exceptional cases it may be necessary to peptonize the milk for a time. At the end of from four to six weeks in ordinary cases, if improvement has occurred, the diet may be increased by the addition of eggs, gruels, cereals, and stewed fruits, and from time to time some fish or green vegetables may be permitted. After the lapse of a month or more, depending upon the condition of the patient, the absolute milk diet should be again resorted to for several weeks; thus alternating, the diet should be continued indefinitely. In general, meat, all highly seasoned and stimulating foods, spices, tea and coffee, etc., should be prohibited, because of their irritating effect upon an already damaged liver; the use of carbohydrate foods and of fats also should be carefully supervised, since carbohydrates not uncommonly undergo fermentation in the intestine and lead to the production of divers toxic substances, and the fats may lead to the formation of fatty acids—all of which may provoke or increase the lesions in the liver. One must individualize, however: in some cases occasional dietetic relaxations, such as the allowance of chicken or other meat once daily or the use of tea or coffee in small or moderate amounts, etc., are attended by mental as well as bodily satisfaction, a more hopeful outlook, and more or less well-sustained improvement.

Comparatively little can be done to influence directly the lesions in the liver, but it is always well to try the effect of potassium (or other) iodide, in the hope that it may do good, and with the knowledge that should the lesions be syphilitic benefit assuredly will follow. Minute doses of mercuric bichloride (gr. $\frac{1}{50}$ to $\frac{1}{100}$ thrice daily), perhaps combined with the iodide, or ammonium chloride, or nitrohydrochloric acid, also seem to do good in some cases. Rolleston warns against the use of arsenic (which has been recommended to stimulate the hepatic cells to compensatory hyperplasia), believing that it may actually cause cirrhosis. The use of the alkaline waters or ammonium chloride perhaps retards the development of the terminal acidosis. The use of organic preparations of liver has proved of no value whatever.

Aside from the foregoing, special attention must be paid to the gastrointestinal tract: indigestion, nausea, vomiting, constipation, diarrhœa, etc., must be prevented as far as possible, and when present should be efficiently treated. In the early stages much benefit may follow a course of treatment at some one of the well-known spas, such as Carlsbad, Marienbad, Vichy, etc. The purgative waters of the spas have a good influence in lessening the intestinal catarrh and the portal congestion, and by the free evacuations that

they induce, ridding the system of noxious autotoxins; their good effect, however, may be as well obtained at home as abroad; or one may give, with the same object in view, sodium sulphate or phosphate, magnesium sulphate, etc., as well as an occasional dose of blue mass or calomel. The good effect of free catharsis, which should be an important part of the treatment throughout the course of the disease, is often seen in diminution in the size of the liver and spleen following copious movements. In some cases in which unusual enlargement of the spleen suggests the imminence of hæmatemesis or other gastro-intestinal hemorrhage, this serious event may be warded off by provoking free catharsis; in other cases ascites may be thus in part absorbed.

The indigestion, nausea, and vomiting are to be treated on general principles; often the disuse of alcohol is followed by immediate improvement in these symptoms; in other cases the additional use of the purgative waters, or saline aperients, is all that is required to secure the desired relief. In other cases, however, medication is required. This should be based, as in catarrhal processes of the gastro-intestinal tract generally, upon the activity of the digestive juices, etc. In the one class of cases, sedatives, such as the different preparations of bismuth, creosote, carbolic acid, dilute hydrocyanic acid, silver nitrate, and hyoscyamus or belladonna, etc., may be desirable; in another class of cases (subacidity) diluted hydrochloric acid, nux vomica, strychnine, and the bitter infusions may lead to the desired result; in still other cases excessive flatulence suggests the use of bismuth, salol, resorcin, creosote, carbolic acid, betanaphthol, and other antifermentatives. Diarrhea should not be indiscriminately checked, since it may serve a useful purpose of elimination; but when severe and obviously exhausting to the patient, it should be met by regulation of the diet and the use of astringents (bismuth, chalk mixture, etc.), and perhaps small amounts of opium. Pain and distress in the region of the liver may be relieved by hot fomentations or a cold compress locally, abdominal massage, the administration of ammonium chloride, and depletive measures.

The occurrence of hæmatemesis calls for treatment similar to that of bleeding in gastric ulcer. The patient should remain in bed absolutely at rest, and tranquillity should be promoted by a hypodermic injection of morphine; an ice-bag may be lightly applied to the epigastrium (supported, if necessary); all food and drink should be withheld; to relieve the dryness and thirst the mouth and lips should be moistened, or small bits of ice may be given to the patient to suck, but the water should not be swallowed. Usually in consequence of the quiet and rest thus induced, and of the relief to the congestion afforded by the bleeding, the hemorrhage ceases of itself. Should it continue, adrenalin chloride (30 minims of the 1 to 1000 solution in one dram of water) may be given by the mouth in the hope that it may reach and influence locally the bleeding point; but neither adrenalin chloride nor other drugs, such as ergot, digitalis, etc., that increase the general blood pressure, should be given hypodermically; in some cases gallic acid, turpentine, aromatic sulphuric acid, etc., by the mouth seem to be of service. If there is much shock and collapse, stimulants and hypodermoclysis should be resorted to. For several days after the hemorrhage no food should be given by the mouth; if deemed necessary rectal enemas may be administered: they serve largely to relieve the thirst; this, if distressing, may also be relieved by water by the bowel. The subsequent feeding and after-treatment is analogous to that of gastric ulcer.

The onset of severe nervous symptoms (chokæmia, acidosis) calls for treatment required in uræmia—free purgation, sweating (by means of hot packs, hot baths, the cautious use of pilocarpine hypodermically, etc.), and diuresis (by means of hot applications to the loins, large amounts of water to drink, alkaline diuretics, digitalis, etc.), and hypodermoclysis. Should diacetic or other acid be present in the urine, large amounts of sodium bicarbonate may be given internally and added to the saline infusion.

The ascites may sometimes be favorably influenced by the use of the purgatives already mentioned, and diuretics, such as the alkaline diuretics, caffeine, sparteine, theobromine, etc.; apocynum cannabinum also has been extolled; sometimes it appears to have the desired result, in consequence of stimulation of the cardiovascular system and the kidneys, but often it induces a distressing enteritis and is extremely depressing. In the majority of cases, however, medicinal measures are of little if any value in ascites, especially if the amount of fluid be large; resort, therefore, must be had to tapping. Tapping may be said to be indicated when the fluid causes local discomfort, dyspnœa, pulmonary congestion or atelectasis, oliguria, etc. There is no advantage in delaying tapping when it has become indicated; indeed, the resort to tapping as often as it may be indicated may lead to the development of adhesions which may augment the already formed collateral circulation. When done under aseptic precautions, the danger of infection may be disregarded; when the fluid is withdrawn slowly there is little likelihood of collapse on the part of the patient. The tapping may be done with a trocar and cannula in the median line midway between the umbilicus and the pubes (after preliminary catheterization of the bladder), or preferably in the linea alba—which has the advantage that the patient may remain recumbent. Continuous drainage is sometimes practised, but does not commend itself to the writer.

Operative relief of the ascites and of the conditions upon which it depends is sometimes undertaken. The operative procedure, which is known as the Talma-Morison operation¹ (omentopexy, epiploexy, Roberts), has for its object relief of the portal circulation, the diversion of the portal blood to the general systemic circulation through the medium of adhesions (and consequent increased vascularity) set up between the liver, the omentum, the parietal peritoneum, etc. How the operation does good has been much discussed. Rolleston points out that the beneficial results cannot be due solely to diversion of the portal blood to the general systemic circulation, since this, imitating an Eck fistula, should tend to provoke or increase toxæmia; he suggests that the benefit attained may be due to: (1) Diminution in the flow of blood through the liver, in consequence of which it is enabled to deal more satisfactorily with the blood that does pass through it, and thus reduces the toxæmia which is probably an important factor in inducing the ascites; and (2) relief of the venous engorgement and a consequent freer supply of arterial blood consequent upon the vascular adhesions over the surface of the liver, as a result of which the nutrition of the liver cells is improved and they

¹ Consult Drummond and Morrison, *British Medical Journal*, 1896, ii, 728; White, *British Medical Journal*, 1906, ii, 1287 (227 cases); Jones, *Transactions of the Medical Society of London*, 1907, xxx, 238; Omi, *Beiträge zur klinische Chirurgie*, 1907, liii, 446 (literature); Lieblein, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, 1908, xviii, 794; and Koch, *Deuxième Congrès de la Société internationale de Chirurgie*, Bruxelles, September 21-25, 1908.

undergo compensatory hyperplasia and become enabled better to perform their function. Of 227 cases collected by Sinclair White, 84 (37 per cent.) resulted in cure and 29 (13 per cent.) in improvement; 34 (15 per cent.) were failures, and death ensued in 75 (33 per cent.). The operation should be done early, while there is hope that sufficient hepatic tissue still remains to discharge reasonable functional demands. The patients submitted to the operation should be carefully selected; associated cardiac, pulmonary, or renal disease, repeated tappings, jaundice, toxæmia, and emaciation should be considered contra-indications.

BILIARY CIRRHOSIS.

Biliary cirrhosis is a chronic disorder of the liver, probably infectious in origin, characterized anatomically by radicular cholangitis and connective-tissue hyperplasia, and clinically by chronic jaundice and enlargement of the liver and spleen. The disorder, now commonly spoken of as Hanot's cirrhosis, hypertrophic biliary cirrhosis with chronic jaundice, in contradistinction to Laennec's, or portal, cirrhosis, was first described by Requin in 1846, and later by Todd (1857) and Hayem (1874); but it was first clearly differentiated by Hanot in 1895. Since then it has been studied especially by Gilbert, Fournier, Castaigne, Chauffard, and others of the French school.

Etiology.—Biliary cirrhosis is especially common during adolescence and early adult life; most cases occur between the twentieth and the thirtieth year; it is rare after the fortieth year, but it is quite common in children (juvenile type), especially in India. In adults about 85 per cent. of the cases (22 of Schachmann's 26 cases) occur in males; in children it is equally common in the two sexes. A seeming hereditary influence is not uncommon; it is mentioned by Osler, Dreschfeld, Finlayson, Rolleston, and others, and it is especially in evidence in the disorder as it occurs in India. Boix, Boinet, and others have pointed out that the disorder may occur in more than one generation of the same family, and that the spleen may be enlarged in certain members of the family who show no evidence of disease of the liver.

The exact nature of the disease is not known. Some, but not all, subjects are alcoholic; and, on the whole, it seems that alcohol plays at most a minor etiological role. In the cases so common in children in India it is not in any way an etiological factor. In other cases, perhaps, it reduces the resisting power of the liver and thus permits of the more ready operation of other causes. There is some rather inconclusive or indefinite evidence that the disorder may be the consequence of infection that provokes a radicular cholangitis; but no definite etiological agent has been discovered, nor are the pathways of infection known with certainty. That the disorder is an infection is suggested by its sometimes developing after infectious diseases, by the common occurrence of attacks of fever and leukocytosis, and the not rare occurrence of general glandular enlargement. Organisms have been isolated from the blood, liver, and spleen during life in several cases, but the evidence is by no means conclusive that they are related etilogically. The postulated infectious agent is believed by some observers to reach the liver by the general circulation, whence the disease is conceived to be a descending cholangitis; by others the infection is believed to be an ascending infection from the duodenum (Rolleston). The concurrent, sometimes ante-

cedent enlargement of the spleen and the occasional occurrence of general enlargement of the lymphatic glands are looked upon as evidence of the widespread activity of some infection; the absence or paucity of dyspeptic phenomena during life and of catarrhal alterations of the duodenum and of the pancreatic duct at necropsy is in favor of the view that the infection does not occur by way of the common bile duct from the duodenum, but by way of the general circulation. Such evidence as is at hand favors the view that the disease is a radicular cholangitis, the consequence of a general blood infection. It seems likely that the poison does not reach the liver by the portal circulation, since portal toxæmia very likely always gives rise to the common or portal cirrhosis; but should the postulated microorganisms of biliary cirrhosis produce a poison in the spleen, which is not improbable, this being conveyed to the liver might induce the lesions of portal cirrhosis, which being added to those of biliary cirrhosis might account for some of the cases of so-called mixed types.

Pathology.—The liver is enlarged and weighs from 2000 to 4000 grams; its shape is maintained, its consistency increased, and its elasticity diminished. The surface is smooth and exhibits very fine granulations (in marked contrast to the coarse granulations of portal cirrhosis); adhesions sometimes occur, but are not very common. The liver is usually dark olive-green in color. The cut surface is dark greenish, or yellowish green in color, smooth, or very slightly granular in advanced cases; usually the liver lobules are more or less obvious, sometimes apparently enlarged, and always separated by widened trabeculæ of connective tissue. The gall-bladder and biliary ducts, the hepatic artery and veins, and the portal vein show no abnormalities, as a rule; gallstones are rarely found, not more frequently than in non-hepatic subjects, which is worth comment in view of the common catarrhal cholangitis which might lead one to expect the formation of bilirubin-calculi calculi.

Microscopically the obtrusive feature consists of an overgrowth of fibrous connective tissue; but the basic and essential process seems to be the radicular cholangitis—which is disclosed by proliferation and desquamation of the epithelium of the small biliary ducts. These lead to obstruction of the lumina of the small ducts, with consequent dilatation of the biliary canaliculi, which in turn are found distended with inspissated bile and bile thrombi (perhaps related to an exudation of inflammatory albuminous fluid). Branching columns of cells, so-called pseudobiliary canaliculi, in and about the periphery of the liver lobules, constitute a conspicuous feature of the lesions. Although by no means limited to, they occur most constantly and in greatest numbers in, this biliary cirrhosis. Some of these formations result from proliferation of preëxisting bile ducts; but some of them undoubtedly are derived from proliferation of the liver cells themselves—which may be looked upon as evidence of reversion to a less highly specialized type of structure, as well as of the histogenetic equivalence of the liver cells and the biliary epithelium. The liver cells otherwise exhibit comparatively inconsequential changes; the retrograde alterations so common in portal cirrhosis are rare; apparently for a long time, much longer than in portal cirrhosis, the liver cells are enabled to maintain their structural integrity. Acute necrotic or autolytic changes, however, may occur at any time and determine the fatal issue. Melchior¹ points out that hyperplastic changes

¹ *Ziegler's Beiträge*, 1907, xlii, 479.

in the liver cells are common, especially in those cases attributed to the influence of alcohol; but some of these cases at least are confused with ordinary portal cirrhosis with an enlarged liver. The new connective tissue occurs in delicate fibrillar bands that not only course between the lobules, but also invade the lobules to a greater or less extent (so-called monolobular, unilobular, or intralobular cirrhosis). In some advanced cases the arrangement of the connective tissue is not so regular; that is, the connective tissue between the lobules becomes disproportionately increased, so that it imitates the so-called multilobular or portal cirrhosis. Perhaps in these cases the multilobular fibrosis is an added feature, and results from the activity of poisons elaborated in the spleen or in the intestinal tract, or from the more common causes of portal cirrhosis, such as alcohol. The conditions under these circumstances are somewhat analogous to those of Banti's disease—primary splenomegaly with terminal cirrhosis of the liver, although, of course, jaundice is not a feature of Banti's disease. The new connective tissue consists in part of elastic tissue, but proportionately less than in portal cirrhosis.

All the organs of the body are bile-stained. The spleen is enlarged, and commonly weighs from 600 to 1000 or 1500 grams or more; in children it may be as large as or larger than the enlarged liver. It is firmer and denser than normally, and often adherent to the adjacent tissues (perisplenitis). Microscopically it reveals lymphoid and endothelial hyperplasia, and fibrosis with consecutive atrophy of the parenchyma. The pancreas usually shows no noteworthy lesions; occasionally a slight periductal fibrosis has been observed. The gastro-intestinal tract also shows no noteworthy lesions, in particular no congestive or catarrhal lesions of the duodenum and the diverticulum of Vater. The lymphatic glands in the portal fissure and usually the adjacent glands are enlarged, œdematous, and congested, but they exert no pressure on the biliary ducts. Occasionally the lymphatic glands in other parts of the body are similarly, although less, affected.

Symptoms.—The onset of the disease is insidious: for a long time there may be ill-defined complaint of poor health, general malaise, weakness, loss of flesh, etc.; or there may be a sense of weight or distress, perhaps actual pain, in the right hypochondrium; rarely there is some dyspepsia, coated tongue, poor appetite, perhaps nausea, diarrhoea, etc.; in a few cases the patient's attention may be first directed to increase in the size of the abdomen.

In the great majority of cases the first obtrusive symptom is jaundice, which is slight at first, not more than a lemon tint to the skin or the conjunctiva. The jaundice varies in degree from time to time, periods of exacerbation and of regression alternating, but on the whole it tends to increase with the progress of the disease, and eventually it may become extreme, usually greenish; sometimes the markedly bronzed color suggests or is mistaken for that of Addison's disease. The usual concomitants of protracted jaundice are seen: there is often intense itching, eczema, lichen, sometimes xanthelasma, etc.; bleeding from the nose or the gums or other mucous membranes may occur from time to time for a long period, or there may be recurring attacks of purpura; the urine shows the presence of bile pigment. The faeces also contain bile pigments and may be very dark (in contrast to the clay-colored stools of obstructive jaundice).

In addition to the jaundice, the disease is characterized by periodic attacks of more or less severe abdominal pain, especially in the region of the liver,

sometimes attended by nausea and vomiting, and associated with fever, leukocytosis, and increase of the jaundice. These attacks occur without regularity and without demonstrable cause, and they last variable periods—several days to several weeks. Usually the fever does not go above 102°; in some cases there is slight fever for long periods. Occasionally these exacerbations are attended by the nervous and other phenomena of icterus gravis, and with high fever, delirium, coma, etc., the patient may die.

The other manifestations of the disease are not characteristic. There may be dyspnoea from impeded action of the diaphragm and pressure upon the lungs and the heart by the enlarged liver and spleen. The myocardium may become disorganized, so that dilatation ensues; the pulse is rarely slowed. Diarrhoea is the rule rather than the exception.

Examination usually reveals an obvious bulging of the lower right costal arch and the upper part of the abdomen, which is found to be due to a much and uniformly enlarged liver; it may reach, in the right nipple line, from the fourth rib, or higher, to near or below the transverse umbilical line; it occupies a proportionate area in the epigastrium; and it extends well into the left hypochondrium. It presents a smooth surface; rarely it may be slightly irregular from fine granulations of the surface (which, however, are scarcely appreciable by palpation) or from adhesions. The organ is firmer than normally, and its edge is sharp and well-defined. Usually the enlargement is progressive, although the size of the organ may vary somewhat from time to time; rarely toward the end some decrease in size may become apparent (usually more apparent than real), attributed to contraction of the newly formed connective tissue or secondary portal cirrhosis. The liver may be moderately but often is not at all tender. The gall-bladder is not enlarged, except in the event of some complication.

The spleen is markedly enlarged, much more so than in portal cirrhosis; it is hard and presents a sharp and well-defined edge. Some writers believe that the enlargement of the spleen may antedate that of the liver. In children the spleen may be actually larger and heavier than the enlarged liver—the juvenile type of biliary cirrhosis (cirrhose biliaire splénomégalyque of Gilbert and Fournier). On auscultation over the spleen one may sometimes hear a soft murmur or friction (perisplenitis).

The blood reveals oligocythæmia, leukocytosis (increased at the time of the periodic exacerbations), diminished coagulability, etc. The urine, in addition to containing bile pigments, is high colored and of increased specific gravity; albumin and sugar are usually absent.

Rarely a bulbous enlargement of the terminal phalanges of the fingers (Hippocratic fingers) and of the toes occurs; it is due to thickening of soft tissues (*x-ray* examination shows no bony overgrowth), and although it resembles the hypertrophic pulmonary osteo-arthritis of Marie, it cannot be correlated with any disease of the lungs; presumably, however, it is due to some unknown toxin, which is not peculiar to biliary cirrhosis, since it may be found in other types of protracted jaundice. Phenomena more significant of synovitis and arthritis are rarely observed. In children growth is stunted, the condition spoken of as infantilism develops, and the onset of puberty is delayed.

Types of Biliary Cirrhosis.—The varying relations between the size of the liver and of the spleen and the time of the onset of these enlargements have led certain French authors, especially Gilbert, Chauffard, Fournier, etc.,

to postulate different types of the hypertrophic biliary cirrhosis: (1) The common type, in which the liver and the spleen are concurrently and equally enlarged; (2) a splenomegalic type, in which the enlargement of the spleen is the predominating feature; (3) a hypersplenomegalic type, in which the spleen is larger than the liver; (4) a metasplenomegalic type, in which the enlargement of the spleen is the antecedent condition; (5) a hepatomegalic (microsplenic or asplenomegalic) type, in which the enlargement of the liver is the predominating feature; (6) a presplenomegalic type, in which the enlargement of the liver is the antecedent condition; and (7) an atrophic biliary cirrhosis. These differentiations are of minor clinical significance, although they suggest that perhaps all cases of biliary cirrhosis are not of the same nature and genesis.

Diagnosis.—The diagnosis, as a rule, presents no unusual difficulties, although it is made more frequently than the rarity of the disease warrants. The important diagnostic features consist of the occurrence in a young, usually non-alcoholic, subject of chronic progressive jaundice, with considerable and persistent enlargement of the liver and spleen, and periodic attacks of abdominal pain, fever, and leukocytosis, with subsequent increase in the jaundice, and no signs of cholelithiasis or portal obstruction.

There should be no difficulty in differentiating the more common or portal cirrhosis—which occurs, as a rule, in older and alcoholic subjects and is attended by more marked dyspeptic symptoms and by evidences of portal obstruction. In portal cirrhosis hemorrhages, especially hæmatemesis, often occur early and are commonly profuse; whereas in biliary cirrhosis hemorrhages are usually a late manifestation and small; they are attributable to disorganization of the blood and to degenerative changes in the bloodvessels, the result of long-continued jaundice and nutritional disorders; they are most common from the oral and nasal mucous membrane and into the skin; and hæmatemesis is rare. Jaundice sometimes occurs in portal cirrhosis, but it is a complication and catarrhal in nature; in biliary cirrhosis it is the conspicuous feature, and, although permanent, it is not associated with absence of bile from the fæces. Constipation is common in portal cirrhosis, diarrhœa in biliary cirrhosis. Portal cirrhosis usually runs a fatal course within three years or less; biliary cirrhosis not uncommonly lasts from five to ten years or more.

The clinical phenomena of biliary and of portal cirrhosis are sometimes combined; that is, jaundice supervenes in a case of portal cirrhosis with enlarged liver and ascites, or ascites develops in a case of supposed biliary cirrhosis with enlarged liver and jaundice. This has led some authors to believe that biliary cirrhosis is not a disease entity; others postulate mixed forms, or the superaddition of portal cirrhosis to the final stages of biliary cirrhosis. The diagnostic difficulties are much simplified by bearing in mind that biliary cirrhosis is a rare disease; that, as already pointed out, although jaundice is not an essential part of portal cirrhosis, it occurs in from 15 to 20 per cent. of the cases, being due to a complicating radicular cholangitis; and that inasmuch as the liver is usually enlarged in portal cirrhosis (although it may become small), an enlarged liver with ascites and jaundice does not necessarily mean a mixed form of cirrhosis, but is quite consistent with the natural history of portal or the common type of cirrhosis. On the other hand, the very rare cases in which ascites supervenes after the long continuance of biliary cirrhosis may be variously interpreted:

in some cases, especially in those without any evidence whatever of portal obstruction, the ascites may be due to disease of the peritoneum (perihepatitis, peritonitis, etc.); in other cases it may be due to gross obstruction of the portal vein, or if terminal to cachectic states; in other cases perhaps the lesions of portal cirrhosis are added to those of the primary biliary cirrhosis.

When the jaundice is slight and perhaps readily overlooked, other forms of enlargement of the liver, such as passive congestion, fatty liver, amyloid liver, leukaemia, malaria, syphilis, and hæmochromatosis must be differentiated. Other forms of chronic jaundice also must be excluded, such as cholelithiasis and carcinoma of the liver and the biliary ducts.

Prognosis.—The outlook as to cure is hopeless. The disease usually runs a protracted course, averaging four or five years, but not uncommonly continuing for ten years or more. The periods of remission of the symptoms often are of long duration and the general health may be well maintained; but the exacerbations may be severe and seriously undermine the general resistance, and they may lead to a severe form of jaundice and death. The prognosis is especially bad in the event of severe jaundice with nervous symptoms (fever, delirium, coma, etc.), recurrent hemorrhages, marked general weakness, intercurrent complications, etc.

Treatment.—The treatment is largely symptomatic, and in general does not differ materially from that advised in portal cirrhosis. The hygienic conditions should be the best obtainable, and should comprise much fresh air, non-exposure to cold and damp, moderate and carefully regulated exercise, etc. The diet may be more generous than in portal cirrhosis, but with a view not to provoke gastro-intestinal derangements; the patient should avoid dietetic indiscretions, alcohol, etc. Constipation should be overcome by the use of calomel, blue mass, and the salines.

The probable infectious nature of the disorder suggests the use of antiseptics, such as urotropin and the salicylates, which are eliminated with the bile. Their use has been followed by some apparent improvement. Good results also are reported to have followed drainage of the gall-bladder and biliary ducts; but the diagnosis in some of the cases is open to question: undoubtedly they may have been cases of infection of the biliary tract, but probably not of biliary cirrhosis.

TUBERCULOSIS, SYPHILIS, AND ACTINOMYCOSIS.

Tuberculosis, syphilis, and actinomycosis of the liver have already been discussed.¹

CARCINOMA OF THE LIVER.

Etiology.—Carcinoma of the liver may be primary or secondary. Primary carcinoma of the liver is a rare disease: among 11,500 necropsies, Hale

¹ *Tuberculosis*, by Dr. W. G. MacCallum and Dr. Lawrason Brown, in volume iii, pp. 232, 266, and 320; *Syphilis*, by Dr. William Osler and Dr. John W. Churchman, in volume iii, p. 478, and *Actinomycosis*, by Dr. James Homer Wright, in volume i, p. 334.

White¹ found only 11 cases. Secondary carcinoma of the liver is more common, the ratio of the primary to the secondary being 1 to 25 (Hale White) or 1 to 40 (Hansemann²). Among 4200 necropsies, Hale White found 136 examples of secondary deposits in the liver, of which at least 126 were carcinomatous. Primary carcinoma is more common in men than in women (6 to 5 of Hale White's 11 cases), and it occurs in adult life and old age; secondary carcinoma is more common in women (the ratio being 4 to 3, Hale White), on account of the greater frequency of carcinoma in women (breasts and genitalia). The causes of carcinoma of the liver are those of carcinoma in general and are as ill understood.

Pathology.—Primary carcinoma of the liver appears under three forms: (1) Nodular or multiple carcinoma. This is the most common form, occurring in 65 per cent. of the cases (Eggel³). The nodules appear as whitish or grayish-white opaque masses, scattered variously throughout the liver tissue; they grow rapidly, frequently become necrotic and consequently umbilicated, and are sometimes the seat of hemorrhagic extravasation. (2) Massive carcinoma is the type occurring in 23 per cent. of the cases (Eggel). It appears as a whitish or grayish-white opaque mass, often occupying almost if not quite an entire lobe (usually the right). It is sharply defined from the surrounding hepatic tissue, which is compressed and atrophic, and sometimes contains smaller secondary nodules. (3) Infiltrating or diffuse carcinoma is the type occurring in 12 per cent. of the cases (Eggel). One or both of the major lobes of the organ become pervaded by new-growth, and often the seat of considerable new fibrous-tissue formation, so that the macroscopic appearances are similar to, and are commonly mistaken for, portal cirrhosis (the cirrhosis with multiple adenoma, or with carcinoma, of Hanot and Gilbert, and other French authors). Microscopically the cells in carcinoma of the liver are usually polyhedral in type, and are believed to be derived from the hepatic cells; rarely they are columnar in type, and are then referred to proliferation of the epithelium of bile ducts. Transitional types also occur, which suggests the histogenetic equivalence of the liver cells and the cells of the biliary ducts. It is likely, as maintained by many writers, Loehlein⁴ lately, for instance, that the growth may develop from either the hepatic or the biliary cells, and that it not infrequently develops from benign hyperplasia. In some cases, therefore, the sequence of events is represented by hyperplasia, adenoma, carcinoma. It is doubtful, however, whether the designation carcinoma with cirrhosis is as widely applicable as maintained by French authors—who believe that this represents one-third of the cases of primary carcinoma of the liver. In perhaps the majority of the cases the condition is merely cirrhosis with considerable hyperplasia of the liver cells (nodular cirrhosis); appearances suggesting carcinoma are sometimes due to softening of some of the so-called "hobnails," with rupture into the hepatic or the portal veins, and to thrombosis—either of which may be the antecedent condition. In the nodular and massive varieties secondary deposits sometimes occur; these are rare in carcinoma with cirrhosis—which also suggests that the condition is not as commonly carcinoma as is often maintained.

¹ *Allbutt's System of Medicine*, 1900, iv, 204.

² *Bert. klin. Woch.*, 1890, xxvii, 353.

³ *Ziegler's Beiträge*, 1901, xxx, 506 (literature).

⁴ *Ibid.*, 1907, xlii, 532 (literature).

Secondary carcinoma of the liver appears usually as multiple, whitish, grayish-white, or yellowish-white nodules scattered throughout the organ, most commonly at the periphery, where they often occasion perihepatitis. Often they become umbilicated, and softened or hemorrhagic. The adjacent liver tissue is compressed, atrophic, and degenerated. Microscopically the growths partake of the nature of the primary tumor.

Symptoms.—These are not notably different whether the growth be primary or secondary, although variations occur and, in perhaps one-half of the cases of secondary carcinoma, symptoms referable to the primary growth are not apparent.

Usually the onset is insidious; the patient may complain of general ill health, languor, anæmia, emaciation, etc., for a long time before the true nature of the disorder is disclosed; in many cases, certainly in at least one-third of the cases of secondary carcinoma, the actual conditions are recognized only at autopsy. In some cases, symptoms referable to the primary growth precede, for a variable time, symptoms significant of involvement of the liver; but in a considerable number of cases, carcinoma of the stomach, rectum, etc., may be for a long time latent; or the symptoms are not obtrusive, and the first noteworthy manifestations may be referable to the liver. In a small percentage of cases the primary growth remains insignificant; the lesions are most marked in the liver, and the symptoms almost, if not exclusively hepatic, consisting of local pain and distress, jaundice, ascites, and enlargement of the liver.

Local discomfort is quite common, but even in cases of very large carcinoma there may be no actual pain. The sense of discomfort and weight is usually proportionate to the size and rapidity of growth of the carcinoma; it is often increased by the left lateral posture, which increases the tension on the ligaments. Actual pain sometimes occurs, and may be quite severe, in which event it is attributable to a concomitant perihepatitis; it often radiates to the right shoulder, and may be increased by motion. Sometimes there is more or less local tenderness, especially on deep palpation and occasionally there is severe colicky pain.

Jaundice is present in more than one-half of the cases; it is variable in degree, at first, at least, depending upon its cause, but it tends to increase and become permanent. It may be due to a variety of causes that are often difficult to differentiate: Thus, it is not infrequently due to compression of the common bile duct by a primary growth in the head of the pancreas, or to secondary nodules in the lymphatic glands; it may be due to primary carcinoma of the biliary ducts; or it may be due to pressure on the intrahepatic ducts by sufficiently large nodules, to extension along the lumen of the biliary ducts, or to more widespread obstruction, such as may occur in massive carcinoma. In the last-mentioned instance, if jaundice occurs, which is unusual, it is slight in grade and is not associated with clay-colored stools. In the other cases the jaundice is obstructive in origin; it increases in intensity until it becomes of the most severe grade; it is permanent, although for a time, at least, it may vary in intensity; and the stools are clay-colored.

Ascites is somewhat less common than jaundice; it occurs in almost if not quite one-half of the cases; both occur together in about 20 per cent. of the cases, in which event the ascitic fluid is bile-stained; otherwise the fluid is pale, of low specific gravity, and contains a small amount of albumin; occasionally it is hemorrhagic or chyliform. It varies considerably in

amount, but is usually moderate and often for a considerable time stationary; it rarely requires tapping. Perhaps in some cases the ascites is due to portal obstruction, but this is unusual, since other evidences of portal obstruction are usually entirely absent or in abeyance. In most cases the ascites is due to perihepatitis or peritonitis; in some cases to carcinomatous involvement of the peritoneum, which is doubtless more common than is suspected.

Progressive enlargement of the liver is the phenomenon that determines the diagnosis. The enlargement may be moderate or very great. Usually it involves the right lobe more than the left, but the percussion dulness is increased in all directions. The liver may be observed to increase notably in size under observation. The edge is usually firm and hard, and it, as well as the palpable surface, is quite irregular—nodular. The nodules vary considerably in size; usually they are firm, especially when small, but as they grow they often soften in the centre and hence become depressed or umbilicated; sometimes this umbilication can be made out by palpation, which is quite diagnostic of carcinoma; in other cases they may seem semi-fluctuating. Sometimes they suddenly increase in size from hemorrhage; occasionally, as they degenerate they become smaller. In the massive carcinoma this nodular formation, of course, does not occur, but one can often make out marked distortion due to a very large new-growth. In advanced stages the irregularly enlarged and nodular liver may be quite apparent on inspection. Palpable and audible friction may sometimes be made out. The gall-bladder can sometimes be palpated as an enlarged pear-shaped tumor at the lower edge of the liver. In a few cases secondary nodules develop along the falciform ligament, and become palpable at or through the linea alba, at the umbilicus; these are conclusive evidence of the real nature of the disorder, but in some cases a microscopic examination of an excised nodule may be required to eliminate tuberculosis.

In many cases the base of the right lung becomes compressed, with dulness and feeble or bronchovesicular breath sounds, etc. In other cases the growth in the liver forms an attachment to, and finally penetrates, the diaphragm, and sets up pleuritis that may become purulent. In rare cases a nodule in the liver penetrates a branch of the hepatic vein, and the carcinomatous cells, being carried to the lung, set up widespread metastasis. Any of these conditions may be responsible for a harsh, dry cough, that is not uncommon; but in some cases no abnormalities can be detected on physical examination to account for the cough, which then must be attributed to irritation of the diaphragm (perihepatitis) or the pleura, etc. The liver sometimes merely by reason of its weight, and in other cases by causing kinking, impedes the circulation in the inferior vena cava, and gives rise to dilatation of the superficial veins of the abdomen (but leaves the region of the umbilicus free—which is a point of distinction from the dilatation due to portal obstruction); in other cases oedema of the feet and legs also results from this cause, but, especially in advanced cases, this may be due also to anæmia, cardiac weakness, hepatic toxæmia, thrombosis of the femoral veins or the ascending vena cava, etc. Albuminuria is occasionally observed late in the course of the disease, and is attributable to anæmia and toxic degeneration of the renal epithelium (hepatogenous albuminuria, Tessier); indicanuria and increase of the nitrogenous extractives also may be observed. Hydronephrosis may ensue from pressure on the ureter or renal pelvis. Gastro-intestinal symptoms are often present, apart from the cases of primary carcinoma of

the stomach; vomiting may result from pyloric obstruction (pressure or kinking). The spleen may become enlarged, especially in the event of pylethrombosis or carcinoma with cirrhosis.

In the course of time the patient loses strength and emaciates (rarely, when the liver increases rapidly in size and attains huge dimensions, the weight may remain stationary or actually increase); he becomes anæmic and cachectic, and phenomena attributed to hepatic insufficiency (cholæmia) develop.

Diagnosis.—This is not always possible, especially in the cases of secondary growth in which the deposits are few in number, small in size, and deep within the substance of the liver. In another series of cases, in which a primary growth is obvious or found on examination, and in which there is persisting severe jaundice, cachexia, and a rapidly enlarging and nodular liver, the diagnosis can often be made on inspection alone, especially if the patient be made to take a deep inspiration (whereupon the irregular nodular liver, covered only by a thin layer of skin, will be seen to descend). Between these extremes lie the majority of cases, in which the diagnosis is often difficult. Should the liver seem to be the seat of disease, one must determine (1) whether a new-growth is present; (2) whether, if present, it involves the liver; and (3) if so, whether it is primary in the liver.

Difficulty is sometimes experienced in determining whether a new-growth is present, since it may be imitated by fæcal accumulation in the colon, and by inflammatory thickenings about the gall-bladder and the biliary ducts. Fæces, however, may be indented and pushed about, they change their position spontaneously from time to time, and they may be removed altogether by cathartics and enemas. In inflammatory thickenings about the gall-bladder there is usually a history of past cholecystitis or cholelithiasis; the mass is in the region of the gall-bladder and is single (that is, there are no other nodules on the surface of the liver); the liver may not be enlarged at all, and if so, the enlargement is slight or moderate and uniform; jaundice, if present, varies from time to time, and may clear up altogether (which is practically unknown in carcinoma); and the patient's general health is not so seriously involved.

One must avoid the error of mistaking tumors of adjacent organs for a new-growth of the liver. This can usually be done by attention to the symptomatology, a careful physical examination, and by a study of the gastric contents, urine, etc. Tumors of other organs, which are near the liver, may have respiratory movement, but such movement is less common in renal and other retroperitoneal growths, which in addition fill out the loin, are covered in front by the intestine, and do not project so high toward the thorax as hepatic tumors. Tumors of the stomach have a significant symptomatology, often cause dilatation of the stomach, characteristically change the gastric contents, and can usually be found quite separate from the liver. New-growths of the omentum, or the thickened indurated omentum of tuberculous or other chronic process, usually has a peculiar elongated or irregular shape and is separated from the liver by a zone of tympany, and the free edge of the liver can usually be felt free and uninvolved.

Should the disorder be definitely located in the liver, one has to eliminate other causes of enlargement, especially cirrhosis, syphilis, abscess, hydatid disease, obstruction of the common duct, as well as other less common disorders, such as passive congestion, constricted liver, chronic malaria, leu-

kæmia, amyloid disease, fatty liver, diabetes, etc. The last mentioned are readily differentiated by attention to the attending clinical phenomena.

In portal cirrhosis there is a history of alcoholism; evidences of portal obstruction are common; the liver is usually uniformly enlarged, although as the disease advances it may grow smaller (in new-growths it continues to increase); the palpable nodules are small, not umbilicated, and not surrounded by intervening areas of smooth liver. Biliary cirrhosis occurs, as a rule, earlier in life than carcinoma; the jaundice is early, moderate in grade, and yellowish (contrasting with the deep greenish jaundice of carcinoma), since some bile always reaches the intestine, the stools are bile-stained (contrasting with the pale, putty-like stools common in new-growths); the liver is uniformly enlarged and smooth; the spleen is enlarged; and the course of the disease is less rapid than in carcinoma.

In syphilis of the liver the pain is often more marked on account of perihepatitis; the liver does not increase so rapidly as in carcinoma, and it lessens rapidly under the influence of antisypilitic treatment; and there is a history or there are signs of syphilis elsewhere. Abscess which is sometimes simulated by those cases in which the carcinomatous nodules soften and become semifluctuating, and in which the fever of cachexia, anæmia, or terminal infection ensues, may be differentiated by the antecedent disorder, by the absence of cachexia, by the less common occurrence of jaundice and ascites, and by the more common hectic fever, etc.

Hydatid disease of the liver is much rarer than carcinoma; it develops, as a rule, earlier in life, and is of slower course; the enlargement of the liver is usually single (perhaps double, rarely multiple), smooth, and not tender; the spleen is enlarged; there is seldom jaundice and rarely if ever ascites; and cachexia does not ensue until the disease has lasted a long time.

Having determined the liver to be the seat of carcinoma, it is often difficult, sometimes impossible, to say whether the new-growth is primary or secondary. In at least one-third of the cases (in 50 per cent. according to Leube) the primary growth is latent; but in some cases in which it is apparently latent it may be detected upon careful study. Attempts to find it should always be made, since, as already said, secondary growths of the liver are from 25 to 40 times as common as primary growths. The presence of a growth in the liver, then, is presumptive evidence of its secondary nature; this is rendered more likely by the presence of many nodules (which, however, do not exclude a primary liver growth with secondary circumferential nodules); by more marked emaciation, due to the multiplicity of the tumors; and by the less rapid course and less rapid enlargement of the liver. Primary growths usually are single and cause uniform enlargement of a part of the liver; they less frequently cause jaundice and ascites; and they grow more rapidly and hence lead to less emaciation, but to earlier death.

Prognosis.—The diagnosis, of course, brings with it the prognosis: the outlook is hopeless. Hale White gives the duration of life as four months from the development of symptoms in primary carcinoma, and seven months after the development of symptoms referable to the liver in secondary carcinoma.

Treatment.—The treatment is purely palliative, and has for its object promotion of the comfort of the patient and relief of his distress. Rare cases have been reported in which a single growth has been removed by operation.

SARCOMA OF THE LIVER.

Sarcoma of the liver may be primary or secondary, but it is very rare.¹ Primary sarcoma may occur as a nodular or a diffuse growth, and may originate from any of the connective tissues of the liver or from the endothelium of the bloodvessels or the lymphatics (angiosarcoma, endothelioma, perithelioma). Secondary sarcoma may follow a primary growth anywhere else in the body, but it is especially common after sarcomas of the bones, adrenal, mediastinum, and uveal tract; the last mentioned are likely to be pigmented. Clinically sarcoma cannot be distinguished from carcinoma, except perhaps in the cases that occur in infants,² in whom a tumor is more likely to be sarcomatous than carcinomatous. Sarcoma of the liver runs a rapid course, and soon terminates fatally.

BENIGN TUMORS OF THE LIVER.

Benign tumors, such as adenoma, angioma, so-called fibroma, lipoma, etc., are occasionally observed, but are chiefly of pathological interest. Adenomas³ may be single or multiple, and may develop from the hepatic cells or from the biliary ducts. The biliary-duct adenoma may become the seat of cystic dilatation (cystadenoma or adenocystoma). Another tumor, often described as an adenoma, arises not from liver tissue, but from aberrant adrenal in the liver. Multiple adenomas are encountered chiefly in association with cirrhosis (so-called nodular cirrhosis); the adenomatous formations are really localized compensatory hyperplasias of the liver cells. It is difficult, if not impossible, to distinguish between hyperplasia and adenoma: there is no sharp line of demarcation, and both may progress to carcinoma. The single adenoma is not susceptible of clinical recognition unless it becomes very large, when it presents more or less outspokenly the symptoms of carcinoma of the liver. The symptoms presented by multiple adenoma are those of the basic condition—cirrhosis.

Angioma is a rare tumor, although it is quite as common in the liver as anywhere in the body. They may be congenital or develop in advanced life; they may be single or multiple, and small or very large. They cannot be recognized clinically. Most of the so-called fibromas and lipomas are not true tumors, and one may well doubt whether such tumors occur in the liver.

CYSTS OF THE LIVER.

Cysts may be parasitic or non-parasitic. The parasitic cysts are due to infection with *Echinococcus granulosus*.⁴ Non-parasitic cysts may occur alone or in association with cystic disease of the kidneys or congenital

¹ Consult Marz, *Centralblatt für allgemeine Pathologie und pathologische Anatomie*, 1904, xv, 433 (literature).

² Consult Pepper, *American Journal of the Medical Sciences*, 1901, cxxi, 287.

³ Consult Wätzold, *Ziegler's Beiträge*, 1906, xxxix, 456 (literature).

⁴ Consult Stiles in this work, vol. i, 576.

anomalies elsewhere. Moscheowitz¹ found that only in 10 of 85 cases was the liver affected alone. There may be one or many cysts. According to Moscheowitz, the cysts, as a rule, are multiple, and most abundant in the zone of the liver just beneath the capsule. They vary in size from those that are microscopic to those that occupy nearly the entire liver. They are equally common in the two sexes, and occur at all ages. They are believed to originate in aberrant bile ducts, in consequence of inflammatory hyperplasia or of retention of fluid the result of congenital obstruction. The diagnosis may be hazarded from the association of an enlarged liver with tumors in the loins (cystic kidneys), or symptoms of chronic interstitial nephritis.

THE GALL-BLADDER AND BILIARY DUCTS.

ANOMALIES OF FORM AND POSITION.

Rarely the gall-bladder may be absent, misplaced, or misshapen. When the gall-bladder is absent, the common bile duct not infrequently presents a circumscribed dilatation (analogue to the gall-bladder); a similar dilatation occasionally develops after operative removal of the gall-bladder. Rarely there is a double gall-bladder with two cystic ducts. Furthermore, the gall-bladder may be misplaced—to the left of the transverse fissure; or it may be embedded more or less deeply in the substance of the liver, and more or less completely surrounded by liver tissue; or the fundus may penetrate the liver substance and present on the anterosuperior surface of the liver. Sometimes it exhibits an hour-glass constriction, aside from the consequences of inflammatory changes. The biliary ducts vary in length. There may be little if any common hepatic duct, due to the unusual length of the right and the left hepatic ducts. The cystic duct may not join the common hepatic duct until close to the insertion into the duodenum. The common bile duct and the pancreatic duct may enter the duodenum separately; sometimes the common bile duct enters with the duct of Santorini. Finally the biliary ducts may be congenitally obliterated.

Congenital Obliteration of the Biliary Ducts.—This is a disorder characterized anatomically by obliteration or atresia of the biliary ducts and biliary cirrhosis of the liver, and clinically by persistent jaundice and so-called cholæmia which proceed to a fatal issue. The disorder was first prominently brought to the attention of the profession by John Thomson,² who, in 1892, collected fifty cases; Lavenson,³ in 1908, collected sixty-two cases. The nature of the disorder has not been definitely determined, at least for all cases. It is likely, as stated by Rolleston,⁴ that several different conditions may give rise to obstruction or obliteration of the large bile ducts in the newborn, such as peritoneal adhesions set up by foetal peritonitis, syphilis, or a mixed form of hepatic cirrhosis with descending and obliteration.

¹ *American Journal of the Medical Sciences*, 1906, cxxxi, 674.

² *Congenital Obliteration of the Bile Ducts*, 1892.

³ *Journal of Medical Research*, 1908, xviii, 61 (literature).

⁴ *British Medical Journal*, 1907, ii, 947.

ative cholangitis (the disease usually called congenital obliteration of the bile ducts). Lavenson supports the view that in most instances the obliteration is due to an anomaly of development, an atresia of the ducts, and that the associated cirrhosis is the result of the ensuing biliary stasis.

Clinically the disorder is characterized by jaundice, which is present at or develops soon after birth, although rarely it may be delayed for several months; it becomes persistent and intense. The stools become acholic, if not acholic from birth; the urine contains bile pigments; the liver and the spleen are enlarged; vomiting often ensues; and hemorrhages occur from the umbilical cord and into the subcutaneous and the submucous tissues. Gradually the infant emaciates; stupor, coma, or convulsions (cholæmia) develop, and death ensues. The course of the disorder is rarely less than a week; usually it lasts for several weeks or several months. The more benign icterus neonatorum may be distinguished usually by the presence of bile in the fæces and by the less-marked jaundice which soon fades. Syphilis may be excluded by the absence of a history of syphilis in a parent, the absence of other evidences of it, and the non-response to antisypilitic treatment.

Rarely a congenital jaundice persists into adolescence or adult life. This type of disorder was first studied attentively by Minkowski,¹ and later by Chauffard² who, separating it from the aforementioned types, believes the essential change to be, not a radicular cholangitis, but a splenohæmolysis—an anomaly in the destruction of the blood influenced by a primary disorder of the spleen.

INFECTIONS OF THE BILIARY TRACT.

Studies of recent years have shown conclusively that the biliary tract is particularly susceptible to, and frequently the seat of, infections. The result of these infections varies with the virulence of the infecting micro-organism and the resistance offered by the individual subject: they may be insidious or frank in onset, acute, subacute, or chronic in course, and slight or extremely severe in character; or they may be entirely latent. The frank acute cholangitis and cholecystitis, the result of infection with virulent bacteria, are usually so obtrusive in the manifestations, as scarcely to escape recognition; when, however, the infection is more insidious in onset and subacute or chronic in course, and the infecting microorganisms of low virulence, the resulting lesions are of such nature and the symptoms so slight, or altogether absent, that they are often ill understood, misinterpreted, and referred to organs other than their real source.

The introduction of microorganisms of low virulence into the biliary tract may be unattended by pathological lesions; this is the more likely to be the case if the ducts are patent, and the flow of bile unobstructed. Comparatively virulent microorganisms also may sometimes be disposed of if the biliary drainage is free and unimpeded, but usually serious and even fatal forms of disease are thus provoked—suppurative cholangitis and suppurative and gangrenous cholecystitis. Between the extremes of mild and

¹ *Verhandlungen des XVIII Congresses für innere Medicin*, 1900, 316.

² *Semaine médicale*, 1907, xxvii, 25.

more or less innocuous lesions and the severe lesions that lead to quick and early disaster lie the great majority of cases of biliary infection.

As in other mucous canals, the immediate result of moderate or low-grade infection of the biliary tract, is the production of a catarrh with the usual inflammatory phenomena—œdema and congestion of the mucous membrane, increased production of mucus, and desquamation of epithelium. If the biliary circulation is free and unimpeded, the results of this catarrh are washed away for the most part, but on account of special local conditions (largely dynamic) they are likely to accumulate, to become accentuated, and to persist in the gall-bladder; in the event of obstruction to the free flow of bile, these are all the more certain to occur. In many cases the lesions thus provoked are entirely latent or without noteworthy symptoms; they may pursue a short course or they may continue for years; and they constitute the important factor in the etiology of gallstones.

The inflammatory disorders of the biliary tract, cholangitis, cholecystitis, and cholelithiasis, thus represent varying manifestations of infection; etiologically, anatomically, and clinically they have much in common; and they occur in varying relationships the one to the others—singly or combined. It is important to bear in mind that the one infectious agent, the typhoid bacillus, for instance, may give rise to cholangitis, cholecystitis, and cholelithiasis concurrently or sequentially; that one of these disorders, cholecystitis, for instance, having developed, may lead to another (cholelithiasis), and that the secondary condition serves to maintain the first; that acute catarrhal cholangitis may become chronic and is usually then associated with gallstones, and that the one or other of these conditions may lead to suppuration within the biliary tract; that it is often difficult clinically to differentiate these disorders; and that the diagnosis to be complete must often comprise more than one of them.

The Infectious Agents.—The bacteriology of infections of the biliary tract has been studied by a large number of investigators—postmortem, at operation, and experimentally. Although considerable interest attaches to post-mortem studies, the results are frequently vitiated by more or less obvious factors, and they cannot be relied upon implicitly unless the examinations are undertaken within a very short time after death. The conditions at operation, however, are quite different and the results much more trustworthy—although in subsiding or long-standing infections the primary infective agent may not be recovered, since it may have died out or have become overgrown by secondary invaders.

At the German Hospital, Philadelphia, we have studied the bacterial cause of the infection of the biliary tract in 240 of the patients operated upon by Dr. John B. Deaver:

	Cases.	Per cent.
Bacillus coli communis was found in	68	28.33
Bacillus typhosus was found in	27	11.25
Staphylococcus pyogenes aureus was found in	7	2.92
Streptococcus pyogenes was found in	1	0.42
Staphylococcus pyogenes albus was found in	2	0.83
Bacillus coli and Staphylococcus aureus were found in	2	0.83
Unidentified bacilli were found in	6	2.50
The cultures remained sterile in	127	52.92

In general these results do not differ materially from those obtained by other observers. Other bacteria, however, have been isolated from the biliary

tract, such as the cholera bacillus, *Bacillus subtilis*, *Bacillus capsulatus aërogenes*, leptothrix, etc. Biliary infections complicating pneumonia and influenza suggest the possibility of the pneumococcus and the influenza bacillus respectively being the etiological agent, but as far as known to the writer these organisms have not yet been isolated from the local lesions. In addition some etiological importance attaches to anaërobic bacteria—which abound in the intestine. A most suggestive study of this entire question has been published by Lippmann.¹

The Pathways of Infection.—The pathways whereby the biliary tract may become infected are: (1) The diverticulum of Vater and the common bile duct; (2) the portal circulation; (3) the systemic circulation; (4) the lymphatic circulation; and (5) directly through the wall of the gall-bladder or the biliary ducts from the peritoneum.

1. *Infection from the duodenum by way of the diverticulum of Vater and the common bile duct* has long been looked upon as at once the most likely and the most common source of biliary infections, but whether with good reason remains to be decided. Although the frequency of *Bacillus coli communis* and of *Bacillus typhosus* in infections of the biliary tract suggests an intestinal source, these bacteria find a direct pathway from the intestine to the liver by way of the portal circulation. Furthermore, whereas the jejunum and the ileum always contain many bacteria, the duodenum when free from food is often bacteria-free; certainly, in health its bacterial content is small and it does not contain the bacteria often found in cholangitis, cholecystitis, etc. It is quite conceivable, however, indeed it is quite likely, that in conditions of disease of the upper intestine, when bacteria are present in the duodenum, the biliary tract may become infected by way of the diverticulum of Vater; doubtless many of the cases of so-called catarrhal jaundice following gastroduodenitis arise in this fashion. But there are at least two important factors opposing a ready ascending infection of the biliary tract: The one, the action of the sphincter of the diverticulum, which has been estimated by Oggi² as exerting a force equal to a pressure within the common bile duct of 700 mm. of water; the second, the influence of the free flow of bile. Indeed, it is doubtful whether infection of the biliary tract ever takes place by way of the diverticulum of Vater in the absence of stasis of the bile. One of the most important factors in preventing such infection is the free flow, that is, the regular periodic expulsion, of the bile—the free flow of the bile rather than the bile itself, since we now know, contrary to former opinions, that the bile is a quite favorable medium for the growth of bacteria.

2. *Infection by way of the portal circulation* is a common source of biliary infection. Definite experimental proof that the bile may become infected from the circulation was furnished years ago by Blachstein³ and Welch,⁴ and their results have since been amply confirmed and amplified, so that there is no doubt that bacteria transported to the liver by the portal circulation may be found in the bile. Under normal circumstances bacteria carried to the liver by the portal circulation are there destroyed by the bactericidal properties of the liver cells; but there is a limit to these bactericidal properties,

¹ *Le microbisme biliaire normal et pathologique*, Paris, 1904.

² Quoted by Naunyn, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, 1905, xiv, 537.

³ *Johns Hopkins Hospital Bulletin*, 1891, ii, 96.

⁴ *Ibid.*, 121.

which when overcome permit the entrance of bacteria into the biliary passages. Adami having shown that under apparently normal conditions bacteria may be found in the deeper layers of the intestine, in the portal circulation, and in the liver, suggests that they invade the portal circulation through the aid of the leukocytes—which are especially active during digestion, carrying foodstuffs, foreign matters, bacteria, etc., between the epithelial cells to the lymphatic radicles and the portal venules. Ordinarily most of the bacteria are destroyed, probably in large part through the bactericidal property of the normal living intestinal mucosa, in part also by the leukocytes, the lymph nodes, and the endothelium of the liver; sometimes, however, they pass through the liver and gain the bile; in other cases, by way of the thoracic duct, they invade the general circulation, and may be found in the liver, kidneys, etc., of apparently normal animals. This is the latent infection of Adami and certain French and German writers.

3. *Infection by way of the systemic circulation* is probably not an exceedingly common source of infection of the biliary tract. Its importance has been enhanced since we have ascertained the frequency of bacteræmia in the great majority of infectious processes—typhoid fever, pneumococcic and pyococcic infections, etc.; and experimental proof that the biliary passages may become thus infected was furnished, as already mentioned, years ago by Welch and Blachstein, and more recently by Doerr. Doerr found that microorganisms injected into the general circulation of rabbits appear in the gall-bladder within a few hours, and that typhoid bacilli and colon bacilli multiply in the gall-bladder and may be recovered therefrom in pure culture even after the lapse of four months. The occurrence of cholecystitis and cholangitis as a complication of general infections, such as influenza, pneumonia, etc., also suggests the likelihood of the infection occurring by way of the general circulation; but one must concede the possibility of the local biliary infection being due to organisms other than those occasioning the primary infection, and that the complicating infection may occur by way of the portal circulation or the diverticulum of Vater. Infection by way of the hepatic artery is a descending infection and operates as does infection carried by the portal circulation—since both circulations commingle at the periphery of the liver lobules; but infected blood carried by the cystic artery may, although probably rarely, lead directly to infection of the gall-bladder without the intermediation of infected bile.

4. *Infection by way of the lymphatic circulation*, as suggested by G. P. Müller, is probably a most infrequent source of infection.

5. *Direct infection through the wall of the gall-bladder* or of the ducts from the peritoneum has been suggested as a possibility; but excluding cases of general peritonitis in which the gall-bladder may participate secondarily and in which the mechanism of the local infection may be quite obvious, it is doubtful if infection of the biliary tract directly from the peritoneum can occur in the absence of adhesions—in which event it is probably an infection by way of the lymphatic circulation. In this connection, however, we must also bear in mind that the adhesions themselves are an evidence of past infection, and that an obvious infection in the presence of old adhesions is much more likely the relighting of an old, latent infection, rather than a new infection transmitted directly through the walls of the gall-bladder or the gall ducts.

The pathways of biliary infection vary with the infecting agent. *Bacillus coli* infections, which must be looked upon as the most common, doubtless occur most frequently by way of the portal circulation. In most cases the liver in full functional activity is enabled to destroy or render innocuous such colon bacilli as may pass the barrier of the intestinal mucosa and be transported to it; but should the physiological activity of the liver become impaired, or should the colon bacilli become of heightened virulence, as happens in inflammatory and ulcerative processes of the intestine, bacilli of attenuated virulence may pass over into the biliary circulation and, being excreted with the bile, set up a low grade biliary catarrh. This commonly passes unnoticed by the patient, but it is one of the most important factors in the etiology of gallstones, as it is also the most important factor in the complications of gallstones. We must admit, however, that in the event of gastro-duodenitis colon bacilli may infect the biliary passages by way of the diverticulum of Vater and the common duct; in this event, the lesions are likely to be more abrupt in onset and more manifest clinically. Typhoid infection may occur by way of the portal circulation, by way of the systemic circulation, and by way of the diverticulum of Vater. The frequency of gall-bladder infections in typhoid fever finds one of its explanations in the three sources whereby the biliary tract may become infected—although the systemic and the portal circulations are the more important.

ACUTE CATARRHAL CHOLANGITIS (CATARRHAL JAUNDICE).

Etiology.—Acute catarrhal cholangitis, perhaps more commonly called catarrhal jaundice, is a common disorder in young adults, although it occurs at all ages; it is more common in men than in women, largely because of men being more under the influence of the exciting causes. The disorder usually follows in the wake of gastro-intestinal catarrh; the causes of the one, therefore, are the common causes of the other: dietetic indiscretions, excessive indulgence in alcohol and other irritants, infectious fevers, etc. Perhaps most cases follow some dietetic indiscretion, such as eating too much food or indigestible or tainted food, or overindulgence in alcohol, etc. These set up gastro-intestinal catarrh, which spreads to the biliary papilla and causes swelling of the mucosa and obstruction to the free flow of bile; this is the basis of the common attacks of so-called biliousness that are attended by some degree of jaundice.

Jaundice is an occasional accompaniment of certain infectious diseases, such as typhoid fever, pneumonia, etc. In some cases it results from spread to the biliary papilla of gastro-intestinal catarrh, that is, the jaundice is obstructive. That, however, in other cases it results from the action of different toxins on the biliary radicles in the liver or on the hepatic cells, is suggested by the comparative rarity of the jaundice as contrasted with the frequency of gastro-intestinal derangements in these infections; by the occasional epidemic occurrence of jaundice; by the likelihood that some cases are mild types of the more severe disorder known as infectious jaundice, Weil's disease, etc.; and by the fact that some cases, mild and apparently catarrhal in nature for a while, may develop into severe types, such as acute yellow atrophy of the liver, etc. In the present state of our knowledge it seems impossible to distinguish clinically between all cases of mild jaun-

dice; some are catarrhal, some toxic from their inception, whereas in others catarrhal in the beginning, the infection may travel up the ducts and involve the finer intrahepatic radicles. Especial interest attaches to typhoid infections, which may be taken as a type, and which will be discussed under cholecystitis.

Catarrhal jaundice may be due also to passive congestion of the duodenum and the swelling of the mucosa thereby induced; thus it occurs in portal cirrhosis, in advanced stages of cardiac disease, etc.; but the jaundice of portal cirrhosis may also be due to radicular cholangitis, and that occurring in advanced heart disease to pressure exerted on the biliary radicles within the liver. Passive congestion renders the patient quite susceptible to the activities of the ordinary causes of catarrhal jaundice. Catarrhal jaundice also may complicate organic diseases of the liver, but the jaundice that occurs in gummas, carcinoma, hydatid cyst, etc., is more commonly due to pressure on the ducts from without than to catarrh. In some cases of supposed catarrhal jaundice no adequate cause can be determined, the disorder under such circumstances being assigned to personal idiosyncrasy, emotional disturbances, exposure to cold and damp, etc.

Pathology.—The lesions consist of congestion, swelling, œdema, and increased production of mucus at the lower end of the common bile duct and of the duodenal mucosa adjacent to the diverticulum of Vater. In some cases the swelling itself is sufficient to obstruct the flow of bile, but in other cases a plug of inspissated tenacious mucus effectually blocks the diverticulum of Vater. The swelling and congestion often subside after death, so that their absence at the necropsy is not trustworthy evidence of their absence during life. All the tissues and organs of the body are bile-stained; rarely the liver may be swollen; the biliary ducts and perhaps the gall-bladder may be distended from the accumulation of bile.

Opportunity to study the lesions in catarrhal jaundice are rarely afforded, since the disease in itself is not fatal. There are many reasons, however, for believing that the cases interpreted as catarrhal jaundice are not all of one type. In many cases, doubtless, the lesions are as described; in others, perhaps, the lower end of the common bile duct, itself not diseased, is obstructed from without by catarrhal changes in the surrounding duodenal mucosa; in other cases the swelling and consequent obstruction occur perhaps higher up in the common bile duct, or in the hepatic duct, or involve the finer intrahepatic branches. It is not unlikely that many cases are as that recently described by Eppinger,¹ who studied at necropsy the lesions in a case of catarrhal jaundice in which death occurred from accident on the eighth day. The significant lesion was hyperplasia of the lymphoid tissue of the mucosa of that part of the common biliary duct that runs in the wall of the intestine; this had led to complete occlusion of the common duct and dilatation of the rest of the biliary system. Eppinger suggests that this lymphoid tissue is analogous to that of the tonsil, and is provided at the end of the biliary ducts as a means of defence against infection from the intestine; that it is subject to attacks analogous to tonsillitis; that epidemics of catarrhal jaundice may be analogous to epidemics of tonsillitis; and that some attacks of jaundice in young persons developing without obvious cause may be due to a so-called lymphatic constitution.

¹ *Wien. klinische Wochenschrift*, 1908, xxi, 480.

Symptoms.—In most cases the early symptoms are those of the provoking gastro-intestinal catarrh: loss of appetite, bad taste in the mouth, coated tongue, foul breath, epigastric distress especially after eating, flatulence, nausea, and perhaps vomiting; there may be constipation, or perhaps diarrhoea from extension of the intestinal catarrh; and the patient complains of headache, vertigo, mental depression, general malaise, etc. Rarely there is an acute onset, jaundice being the first symptom noticed; but in most cases after the mentioned symptoms have lasted for a few days to a week, the patient notices, or his attention is directed to the fact, that he is slightly jaundiced. The jaundice is of slow and insidious onset, and gradually increases. It is first and best seen in the sclerotic coats of the eyes; soon the integument of the face becomes discolored (especially obvious in blonds), and then that of the rest of the body and the visible mucous membranes. With the appearance of the jaundice, sometimes before there is any obvious jaundice, the stools become pale in color and soon are devoid of bile pigment; the urine becomes scanty, dark in color, deposits an abundant sediment, and shows the presence of bile pigments. Usually the bile pigments can be detected in the urine before the appearance of the jaundice in the sclerotics.

When the disorder is fully developed, that is, at the end of four or five days, the discoloration of the skin and mucous membranes is usually well-marked, but even when highly developed the color is distinctly yellow, the dark olive-greenish discoloration of malignant disease never developing. Pruritus is often marked; sometimes it even precedes the development of the jaundice. The itching is sometimes so extreme as to induce severe eczema, in consequence of uncontrollable scratching. The pulse usually is slowed, 60 or less per minute, and may reveal the diastolic of low vascular tension; sometimes this is obvious as a well-marked capillary pulsation. The patient usually is irritable or melancholic, often sleepy, and he loses flesh and strength.

In most cases neither the liver nor the gall-bladder is palpably enlarged or tender; sometimes, however, the liver can be felt below the costal margin; in cases in which the liver or gall-bladder is much enlarged one should suspect a complicating cholecystitis or inflammation of the intrahepatic ducts. The spleen may be enlarged. Fever (101°) is occasionally present, but it is rarely due directly to the cholangitis, except in the epidemic infectious cases; that which sometimes occurs at the beginning of the attack and lasts a day or two is probably due to the gastro-enteritis; when conspicuous or when it lasts more than several days, it suggests some one of the types of toxic or infectious jaundice (of which, in reality, it may be a mild manifestation). Often late in the disorder the temperature is subnormal.

At the end of about a week, sometimes not until later, the evidences of gastro-intestinal irritation subside, the bad taste in the mouth and the coated tongue disappear, and the appetite returns; in some cases, however, nausea and vomiting persist for ten days or more; and in most cases constipation continues as long as the fæces are clay-colored. Gradually the evidences of biliary obstruction lessen, bile pigments appear in the fæces, and disappear, first from the urine and then from the skin, where, however, traces may be found for several months. Restoration of the general health is often much delayed; although the appetite may return at the end of a week or two, improvement in the general health does not occur until the bile

pigments begin to be removed from the tissues. Ordinarily an attack lasts three or four weeks, but sometimes six weeks or more.

Diagnosis.—In most cases the diagnosis is readily made from the youth of the patient, an antecedent gastro-intestinal catarrh provoked by some more or less obvious cause, and the absence of local pain and of serious involvement of the general health (emaciation, etc.); the diagnosis is further confirmed by the benign and favorable course of the disorder, which in many cases definitely determines the diagnosis. Doubt regarding its catarrhal nature should always be awakened by the occurrence of jaundice in a middle-aged or elderly person, if it becomes deep or olive-tinted, and if it lasts more than six weeks. Jaundice in a middle-aged or elderly person is much more likely to be due to impaction of a gallstone, or to carcinoma compressing the common duct. In gallstones there is usually a history of past infection of the biliary tract, or of definite attacks of cholelithiasis, and the jaundice is commonly preceded by pain; in impaction of the calculus in the common duct there are usually periodic attacks of chills, fever, and sweats, and variation in the degree of the jaundice; occasionally, however, in protracted catarrhal jaundice, fluctuations in the intensity of the jaundice occur, but these relapsing cases are usually referable to repeated dietetic or other indiscretions that may have provoked the attack. Mild grades of so-called toxic or infectious jaundice may be mistaken for catarrhal jaundice—perhaps often they are only different grades of the same process; but in infectious jaundice there is usually an acute onset with fever, and signs of general infection are more prominent—enlarged spleen and liver, albuminuria, etc.

Prognosis.—As a rule, the prognosis is good; the disease of itself does not endanger life, and recovery is usually complete and lasting. Most cases last from three to four weeks, but sometimes longer; cases lasting five months and followed by recovery have been reported, but protracted as well as relapsing cases of jaundice are usually due to causes other than catarrh. So-called abortive cases of slight grade and short duration may subside entirely within two weeks or less. In cases that begin with much fever the prognosis at the beginning should be guarded, since they may be the early stage of, and progress to, serious and fatal forms of jaundice. Protracted cases in middle-aged and elderly persons are sometimes of serious import—in consequence of impairment of the general nutrition and the tendency to hemorrhage. Usually complete recovery ensues, but sequels are not unknown; recurrences are not uncommon, especially in those who repeat the dietetic and other indiscretions provocative of the primary attack. In some cases a mild grade of chronic catarrh persists, which, involving the gall-bladder and the intrahepatic ducts, may lead to hydrops of the gall-bladder, a mild chronic cholecystitis, cholelithiasis, etc., and perhaps also to some case of Hanot's biliary cirrhosis; and from progress of the infection, to infection and chronic inflammation of the pancreas.

Treatment.—The treatment is essentially that of the gastro-intestinal catarrh, commonly provocative of the cholangitis. The patient should go to bed; for several days at least the diet should be reduced to a minimum, and should consist of a moderate amount of milk, preferably skimmed milk or broth. The common distaste for food is a good indication to give no food whatever for a day or two, and to be content to relieve the thirst with copious draughts of water, which are usually relished and well borne. The natural alkaline waters are especially serviceable; but alkalis, especially sodium

bicarbonate, may be added to ordinary water. Nausea and vomiting, if they persist after withdrawal of the food, may be relieved by lavage with hot alkaline water, the drinking of hot alkaline water, or the administration of bismuth subnitrate or subcarbonate, carbolic acid, dilute hydrocyanic acid, cerium oxalate, etc.; or by warm applications to the epigastrium. The bowels should be opened freely. Custom sanctions the use of calomel, which may be given in a single large dose or in repeated small doses (a blue pill is equally efficacious); it should be followed by a saline aperient. These are much more efficacious if taken hot (or in hot water), in which event they exert an increased beneficial action on the gastro-intestinal catarrh. They should be given in sufficient dose to cause one or two free evacuations daily; more are undesirable. The diarrhoea sometimes present in the early days of the attack rarely demands treatment. Usually it subsides spontaneously upon the adoption of a proper dietary regimen and cleansing of the bowels with calomel or blue mass and a saline aperient. Occasionally it may be necessary to administer astringents and antifermentatives, but preparations of opium should be avoided.

After the lapse of several days, when the gastro-intestinal catarrh begins to subside, the tongue to clean, and the appetite to return, additions may be made to the diet, but for some days still, only milk diluted with lime water or Vichy water, thin or strained soups, milk toast or dry toast, fish, etc., should be allowed. Later one may add chicken, chops, rice and other carbohydrates, the finer green vegetables, etc.; but for some time all highly seasoned food and food likely to undergo fermentation must be avoided. The indication of progress toward recovery and of permissible liberality in diet is found not in the degree of jaundice, but in the return of bile pigment to the faeces and its diminution in the urine. The use of the alkaline aperients should be continued. Injections into the rectum of cold water are sometimes very efficacious in stimulating intestinal, and perhaps also gall-bladder, peristalsis. As the biliary stasis begins to subside, diluted nitro-hydrochloric acid and bitter tonics render excellent service. Excessive flatulence is sometimes distressing; it may be relieved by the use of bismuth salicylate, soda-mint, aromatic spirit of ammonia, chloroform water, and antifermentatives; magnesia or rhubarb with sodium bicarbonate and charcoal also are of much service, and relieve the constipation.

The pruritis is best relieved by the measures that tend to relieve the biliary catarrh and thus promote the removal of bile pigments from the tissues. Temporary relief may also be obtained by the use of cold sponging, or of a carbolic acid lotion (1 to 40 or 60), or the internal use of the bromides or other nerve sedatives.

The general health requires attention for some time, often months, after an attack of catarrhal cholangitis. Attention must be paid to avoiding the factors answerable for the primary attack, especially overeating, highly seasoned food, alcohol, etc. Much benefit may be derived from treatment at one of the well-known spas, such as Carlsbad, Vichy, Marienbad, etc.

SUPPURATIVE CHOLANGITIS.

Etiology.—Suppurative cholangitis results from virulent bacterial infection of the biliary ducts. The common infectious agents are *Bacillus coli communis*, staphylococci, streptococci, pneumococci, *Bacillus typhosus*, the

comma bacillus, *Bacillus aerogenes capsulatus*, etc. *Bacillus coli communis* is sometimes found alone, but it is not improbable that in some of the cases, at least, it is a secondary invader, or has been present in association with and has outgrown the other or primary infectious agents. *Bacillus aerogenes capsulatus* usually invades the tissues only during or just prior to the death agony, but occasionally it is the primary infectious agent and may be found during life.

The infection in suppurative cholangitis may occur by way of the portal or the general systemic circulation (descending infection), or by way of the duodenum and the diverticulum of Vater (ascending infection). Infection sometimes occurs in certain general infections, but it is rendered much easier of accomplishment by local disease of the biliary tract; indeed, in the absence of very virulent infection it is doubtful if infection of the biliary tract ever occurs in the absence of local biliary disease, since, as is well known, the free flow of bile usually is an effective preventive of biliary infection. Suppurative cholangitis, however, is occasionally observed in association with general infections. The provoking microorganism, aside from the typhoid bacillus, is often not that of the primary infection, but the colon bacillus or the pyogenic cocci. Usually they reach the biliary ducts by way of the portal or the general systemic circulation, although in the cases in which intestinal lesions occur, such as typhoid fever, and in the event of gastro-intestinal catarrh in pneumonia, influenza, etc., one must admit the possibility of infection by way of the duodenum. This cholangitis rarely occurs without concomitant cholecystitis.

In the great majority of cases suppurative cholangitis is definitely related to local disease of the biliary tract. The local disease acts as the necessary predisposing agent; this comprises all disorders that interfere with the free and unimpeded circulation of the bile (that is, cause stagnation), and reduce the resistance of the biliary ducts. The commonest antecedents of suppurative cholangitis are gallstones and tumors obstructing the ducts. Gallstones themselves are a consequence of low-grade biliary infection and catarrh, and by the obstruction to the flow of bile that they induce, as well as because of the consequent reduction in the resistance of the biliary mucosa, they participate in the formation of a vicious circle. Suppurative cholangitis thus may supervene upon long-standing chronic catarrhal cholangitis with or without cholelithiasis, upon acute impaction of a gallstone in the ducts, and upon chronic infective cholangitis with a gallstone in the common duct (intermittent hepatic fever). Tumors of the biliary ducts or of the adjacent tissues that may obstruct the ducts also favor the development of suppurative cholangitis by obstructing the flow of bile and causing secondary dilatation and reduction in the resistance of the ducts, and, in the event of the growths becoming ulcerated, by affording a means of ready access for bacteria.

There are other rarer causes of suppurative cholangitis, such as the spread to the biliary ducts of infection from hepatic abscesses of other origin; hydatid cysts compressing or rupturing into the biliary ducts; compression of the ducts by an aneurism of the hepatic artery; infection of the ducts by round-worms and liver flukes which may carry infectious bacteria, or by inducing stagnation of the bile favor bacterial infection.

Pathology.—Although the process may be more or less limited, it is usually widespread. The biliary ducts are dilated (in advanced cases often enormously dilated) and filled with purulent material, commonly stained with

bile. The walls of the ducts are much thickened, softened, and obviously the seat of purulent infiltration; the mucosa is congested, oedematous, and covered with a layer of mucus, and in fairly well-advanced cases irregularly ulcerated. Usually analogous changes are found in the gall-bladder. About the terminal branches of the biliary ducts there are usually small abscesses; these may be very small and very numerous, but in advanced cases some at least have attained considerable size, in consequence of confluence of several or many small abscesses. Occasionally there are only a few, rarely a single large abscess. The liver is enlarged, swollen, softened, and opaque; the surface usually is irregular, in consequence of the projection of many small abscesses. The cut surface is quite characteristic: more or less, sometimes enormously, dilated biliary ducts, filled with pus (multiple abscesses), with the intervening liver tissue, the seat of marked periductal congestion, parenchymatous degeneration, and necrosis. Not uncommonly there are smaller abscesses circumferentially about some of the infected intrahepatic ducts, possibly due to transport of infection by way of the lymphatics. The abscesses on the surface of the liver may extend to the adjacent peritoneum and set up perihepatitis or more widespread peritonitis, and perhaps extend to the pleura and the lungs or the adjacent intestine; or the process may involve the portal vein, or the hepatic artery; or the suppuration may extend along Wirsung's duct and induce suppurative pancreatitis and abscess in the lesser omentum. The adjacent lymphatic glands, especially those in the portal fissure, are congested, swollen, softened, and sometimes necrotic. Microscopically the lesions are those common to suppurative inflammation—congestion, oedema, cellular proliferation and infiltration, liquefaction necrosis, etc. The purulent infiltration and necrosis are sometimes so extreme that the structures of the liver no longer can be identified. Because both may be extensively involved, it may be quite difficult to determine whether the process originated in the biliary ducts or the portal vein. In long-standing cases of chronic infection there is more or less pericholangitis fibrous hyperplasia. The liver cells show parenchymatous and fatty degeneration, and here and there small collections of round cells (beginning abscesses unconnected directly with the biliary ducts), or foci of more or less complete necrosis.

Symptoms.—The symptoms are not always distinctive; sometimes not even suggestive. Usually there is a history of antecedent cholelithiasis or vague symptoms that may be interpreted as due to gallstones; in other cases there is some impairment of the general health, which, in the light of subsequent events, may be ascribed to malignant disease; in still other cases the patient suffers from, or has recently suffered from, some general infection, such as typhoid fever, pneumonia, etc. The onset is often insidious; occasionally the first noteworthy symptom is slight jaundice, but in perhaps most cases, especially in cases in which there has been antecedent jaundice due to other causes, the onset of the disease is announced by constitutional phenomena, such as chills, fever, and sweats. These are usually severe, more or less frequently repeated at irregular intervals, and soon lead to considerable impairment of the general nutrition, emaciation, loss of appetite, nausea, perhaps vomiting, diarrhoea, etc.; variable afebrile periods, however, are not uncommon. Jaundice is almost always present, but it is variable in degree; usually it is slight or moderate only, except in cases in which the cholangitis is associated with other conditions that cause intense jaundice,

in which event the jaundice is due not so much to the suppurative cholangitis as to the antecedent disorder. The minor grade of jaundice in suppurative jaundice, as contrasted with the more severe grade in catarrhal jaundice, probably finds its explanation in the more marked disorder of the liver cells and the greater interference with their function, as well as in inflammatory obstruction of the lymphatics, by which, in other types of jaundice, the bile enters the circulation. Rare cases of suppurative cholangitis without jaundice have been described.

Sometimes there is no pain in the region of the liver. Usually there is at least dull aching or discomfort (tension of the liver capsule); now and then there is more severe pain due to acute cholecystitis, cholelithiasis, obstruction of the ducts by tumors or worms, perihepatitis, peritonitis, etc. The liver is enlarged and usually continues to enlarge under observation. It presents a smooth surface, except in the event of an unusually large abscess presenting on the palpable surface; and it is tender; perhaps, also, there may be a tender spot posteriorly on the right side on a level with and 2 to 3 cm. from the twelfth thoracic vertebra (Boas). Usually the gall-bladder is enlarged and tender (acute cholecystitis). The spleen also may be enlarged. Examination of the blood reveals polynuclear leukocytosis, more marked during and just after the febrile periods; sometimes it is absent during the afebrile periods.

Extension of the lesions locally may lead to the ordinary signs of local or general peritonitis, pleuritis, etc., with perhaps evacuation of pus through a bronchus. Absorption of toxins may induce severe headache, joint pains, swelling, and tenderness, and their excretion may be attended by albuminuria and casts. Involvement of the hepatic vein may lead to multiple abscesses in the lungs, infectious endocarditis, general septicopyæmia, etc. Sometimes a single abscess ensues, and may establish communication with the intestine or pleura and lung.

Diagnosis.—The diagnosis is usually apparent from a history of antecedent gallstones or other disorder of the biliary tract, or of typhoid fever or other infection, and the development of chills, fever, and sweats, and jaundice, local pain, progressive enlargement of the liver, perhaps also of the gall-bladder and spleen, and leukocytosis. In the absence of a history of antecedent disease of the biliary tract, the other symptoms mentioned, if at all outspoken, suffice for the diagnosis; but in many cases the symptoms are added so insidiously to those of the antecedent disease, or they are, in the beginning at least, so mild and evanescent, that it is difficult if not impossible to say just when the suppurative process began or how severe it is. A slight and temporarily increased obstruction to the flow of bile may lead to increased absorption of toxins from a chronically infected biliary tract and increase of the general and local symptoms; release of the obstruction may permit of the free discharge of pus from the biliary tract with subsidence of the symptoms. Thus in some cases recurrences and remissions of the symptoms occur, for instance in the discharge of an impacting gallstone, drainage may become very free and lead at least to temporary subsidence of all the symptoms.

Difficulty may be experienced in differentiating suppurative pylephlebitis, other forms of abscess (especially solitary or tropical abscess), malaria, etc. Jaundice is not an essential symptom, but it is suggestive of, and occurs earlier in, suppurative cholangitis than in pylephlebitis or single abscess of the liver. Enlargement of the spleen, while it occurs in suppurative cholangitis and pylephlebitis, is more common in pylephlebitis; but cholangitis and

pylephlebitis may co-exist. In tropical abscess there is usually a history of antecedent dysentery. Malaria may be excluded by the absence of periodicity of the chills, fever, and sweats, the absence of plasmodia from the blood, and the presence of leukocytosis.

Prognosis.—The prognosis is decidedly unfavorable, but it is modified by a number of attending phenomena and the treatment. In view of the difficulty of determining accurately the time of onset of the condition, it is difficult to say how long it lasts; but the acute cases which pursue a progressive course, seldom continue more than three weeks. In less severe cases, in which the infection is less virulent and becomes subacute or chronic, or in which the lesions gradually subside and perhaps become localized to the gall-bladder, or in which a fistulous communication is established with the hollow abdominal viscera or the thoracic viscera, and the pus is discharged, etc., the duration may be much longer and recovery may ensue. The prognosis is materially improved by the spontaneous or operative removal of the source of obstruction (gallstones, worms, etc.), and by the consequent establishment of free drainage.

Treatment.—Prevention of the disorder is much more readily effected than cure. Even minor grades of biliary infection, such as the acute and more chronic catarrhal cholangitis (jaundice), gallstones, etc., should be carefully and persistently treated. This, especially in the event of gallstones, comprises surgical intervention—which not only is curative of the gallstones and the accompanying catarrh, but also preventive of the much more serious suppurative cholangitis.

When suppurative cholangitis has developed, the treatment is essentially surgical, and consists in the establishment of free drainage. Medical treatment may be used in conjunction with surgical, and consists in the use of salicylates, urotropin, and other remedies to disinfect the biliary tract and promote the free flow of bile. Otherwise, medical treatment is purely palliative and analogous to that laid down for acute catarrhal jaundice.

CHRONIC CATARRHAL CHOLANGITIS.

Etiology.—Chronic catarrhal cholangitis may involve only the larger, especially the extrahepatic, ducts, or more or less of the entire biliary tract, or it may be confined to the finer intrahepatic ducts (radicular cholangitis or angiocholitis). Usually the lesions are widespread, involving the larger extrahepatic ducts, more or less of the larger intrahepatic ducts, and the gall-bladder. This form of the disease is due to the factors that provoke acute catarrhal cholangitis, and may result from repeated attacks of acute catarrhal cholangitis, or from chronic low-grade infections. It is most commonly found in association with gallstones, with which its etiology and pathogenesis are so intimately associated that it will be discussed therewith; but chronic non-calculous cholangitis also occurs, being found in association with chronic gastroduodenal catarrh, after certain infections, in association with chronic obstruction, and in association with certain chronic disorders of the liver, such as hydatid cysts, chronic abscesses, etc. Chronic catarrhal radicular cholangitis or angiocholitis is rarely an independent affection.

Pathology.—The lesions of chronic catarrhal cholangitis (of the larger ducts) consist of congestion and swelling of the mucosa, increased production of mucus, round-cell infiltration, and proliferation of the submucosa and

muscularis, with the production of new fibrous connective tissue; these lead to thickening and induration of the walls of the ducts and to pericholangitis.

There may be partial or complete obstruction of the ducts. In the event of complete obstruction, the ducts become enormously dilated (up to the size of a thumb or larger); the gall-bladder and some of the intrahepatic ducts also become dilated. The contents of the biliary tract is usually a clear, almost colorless, often sterile mucus; when the mucus is sterile, the mucosa of the biliary tract is usually quite smooth. In the event of incomplete obstruction the ducts and especially the gall-bladder are less dilated, the contents are usually turbid and often contain bacteria, and the mucosa may be more or less ulcerated.

Symptoms.—The symptoms are those of chronic relapsing jaundice. Rarely this may represent a direct continuation of an acute catarrhal jaundice, being more insidious in onset, and subject to exacerbations and remissions. The attending phenomena are those of acute catarrhal cholangitis—prolonged. In the cases of complete obstruction, the contents of the biliary tract being sterile, there is usually no fever, and remissions in the jaundice are slight. In the cases of incomplete obstruction, remissions in the jaundice are more marked, and the contents of the biliary tract being infected, attacks of chill, fever, and sweats are common. These attacks by no means warrant a diagnosis of suppuration of the biliary tract, although they are evidences of infection. This chronic catarrhal cholangitis may lead to suppurative cholangitis (of which it is often only a minor manifestation), acute infectious jaundice, acute yellow atrophy of the liver, etc.

Diagnosis.—In cases of persisting jaundice that begin seemingly as acute catarrhal cholangitis, one must attempt to eliminate cholelithiasis and malignant disease obstructing the ducts. Often it is quite impossible to differentiate non-calculous from calculous cholangitis with a gallstone in the common duct; attempts to do so are usually only of academic interest. A history of antecedent gallstone colic is suggestive but by no means conclusive. Hypertrophic biliary cirrhosis may be eliminated by the absence of marked enlargement of the liver and spleen.

Prognosis.—The prognosis is dependent upon the associated lesions.

Treatment.—The treatment is essentially that of acute catarrhal cholangitis. In protracted cases operation should be undertaken with a view to drain the biliary ducts; this is the more urgently demanded in view of the impossibility often of excluding obstruction due to a gallstone.

ACUTE CHOLECYSTITIS.

Etiology.—It is largely artifice that leads to a distinction between cholangitis and cholecystitis; the one like the other is an expression of infection of the biliary tract. In some cases the infection may remain confined to the ducts or the gall-bladder. In other cases, developing in the one or the other, the process may extend to that not primarily involved; and it may persist in the gall-bladder and subside in the ducts, or vice versa. A separate discussion of the two conditions seems warranted, however, because cholecystitis is characterized by certain definite and characteristic anatomical and clinical features that develop largely because the gall-bladder is a closed sac with a small and readily obstructed exit, and because a common sequence

of cholecystitis is cholelithiasis, which, having developed, serves to keep active or readily induces cholecystitis.

Like cholangitis, cholecystitis results from bacterial infection, which is facilitated by any cause that reduces the resisting power of the gall-bladder or interferes with the free flow of bile. Mixed infections are not uncommon; and in some cases one organism having set up the lesions may die out spontaneously or be overgrown by another, whence in some cases the gall-bladder contents are sterile or the primary infectious agent eludes detection. The infectious agents reach the gall-bladder usually by way of the portal circulation or the general systemic circulation, although, of course, they may reach it by way of the diverticulum of Vater and the common bile duct. *Bacillus coli* infections doubtless occur most frequently by way of the portal circulation, although in some cases they may occur by way of the diverticulum of Vater; typhoid infections may occur by way of the portal circulation, the general systemic circulation, and the diverticulum of Vater, the portal and general systemic bacteræmia doubtless explaining the frequency of infection of the gall-bladder in typhoid fever; pneumococcic, staphylococcic, streptococcic, influenzal infections, etc., are probably in many cases general systemic infections, although in some cases the infection may occur by way of the diverticulum of Vater; the subject, however, requires more study. Heretofore, for obvious reasons, we have had to be content with a diagnosis of cholecystitis or cholangitis, as the case may be; a noteworthy advance in diagnosis would be the recognition of the etiological agent. When cholecystitis or cholangitis complicates typhoid fever, pyococcic infections, etc., a natural inference is that the microörganismal cause of the general or primary infection is the cause of the local complication in the biliary tract—an inference usually but not always borne out by the fact. A biliary infection complicating or occurring sequentially to pneumonia, influenza, etc., may be due not to the specific cause of the general infection, but to pyogenic cocci or the colon bacillus, the original infectious agent or its toxin perhaps having prepared the way for the secondary invader. Certain zoöparasites also (especially round-worms, bilharzia) may invade the biliary tract, and reaching the gall-bladder may set up cholecystitis and subsequently cholelithiasis; in some cases, however, the zoöparasites act only as the carrier of other infectious agents.

Typhoid Infections.—Major importance and interest attaches to typhoid infections of the biliary tract.¹ It is now definitely known: (1) That the typhoid bacillus is regularly present in the gall-bladder, and commonly in pure culture, in practically all cases of typhoid fever; (2) that the typhoid bacillus may persist in the gall-bladder, as well as within gallstones, years after the patient has recovered from an attack of typhoid fever; (3) that cholangitis and cholecystitis are by no means infrequent complications of typhoid fever; and (4) that a history of antecedent typhoid fever may be obtained in many patients with cholecystitis and cholelithiasis.

The manifestations of typhoid infections of the biliary tract are various:

1. *Acute cholecystitis* occurring during the course of or after typhoid fever does not vary materially from acute cholecystitis due to other bacterial causes, although less likely to become suppurative.

2. The development of *gallstones*.

¹ Consult *Infections of the Biliary Tract*, by A. O. J. Kelly, *American Journal of the Medical Sciences*, 1906, cxxxii, 446, 744.

3. *Primary typhoid cholecystitis*, often associated with cholangitis; that is, an infection of the biliary tract with *Bacillus typhosus* without other evidence of past or present typhoid infection. These cases are probably not uncommon, and are of much interest; the lesions are often very serious. Much allied to these are many of the cases of jaundice that give a positive Gruber-Widal reaction with *Bacillus typhosus*. Since the frequency of primary typhoid cholecystitis and cholangitis has been recognized, and since it has been determined experimentally that the bile has little if any tendency to cause agglutination of typhoid bacilli, these cases of jaundice giving a positive Gruber-Widal reaction are properly interpreted as examples of typhoid cholangitis and cholecystitis; and the serum reaction is to be looked upon as of much value in determining the true nature of the jaundice. There is no doubt that we have much to learn of the true nature of certain ill-understood forms of jaundice, infectious jaundice, Weil's disease, etc. The resemblance that many of these cases bear to typhoid fever has been frequently commented upon, and it has, in fact, been suggested that Weil's disease is in reality a modified form of typhoid fever. Possibly in many cases it is only typhoid (or paratyphoid) infection of the biliary tract. To determine their true nature bacteriological investigations of the blood and the faeces, as well as a study of the Gruber-Widal reaction, should be undertaken in all cases of jaundice; positive serum reactions have been reported in dilutions as high as 1 to 1800.

4. *Chronic Carriers*.—In many cases the typhoid bacillus may be recovered from the gall-bladder years after an attack of typhoid fever (fifty-two years, Gregg). The continuous re-infection of the intestinal tract by the frequent discharge of virulent typhoid bacilli from a chronically infected gall-bladder may or may not be of much significance to the individual, depending upon the degree of immunity developed, but it is of much significance from an epidemiological point of view, since there is no doubt that these apparently healthy harborers of typhoid bacilli spread the infection and give rise to more or less extensive epidemics.¹

Although bacterial infection is the essential factor in provoking acute cholecystitis, other factors are of contributing or predisposing importance; these are such as (1) reduce the vitality and resisting power of the gall-bladder and biliary ducts, and (2) interfere with the free flow of bile. Among the first mentioned are: trauma, such as blows and other injuries in the region of the gall-bladder, which rarely may reduce the resistance of the gall-bladder, and render it vulnerable to bacteria already present or that may be brought thither; a previous attack of cholecystitis and gallstones, which favor infection; local diseases of the intestinal tract which may act as the source of the bacteria; and general disorders, such as Bright's disease, in which complicating or terminal infections are common. Among the conditions that act as predisposing causes by interfering with the free and unimpeded flow of bile are gallstones, usually associated with chronic cholangitis, tumors or foreign bodies (worms, etc.) within the ducts, tumors without the ducts, kinks or cicatrices compressing the ducts, etc.

¹ Consult Blumenthal, *Deutsches Archiv für klinische Medizin*, 1906-7; *Journal of the American Medical Association*, 1908, li, 1986; Park, *ibid.*, 1908, li, 981; Forster, *Münchener medizinische Wochenschrift*, 1901, i; Scheller, *Zentralblatt für Bakteriologie*, 1908, 385; and a discussion, *Verhandlungen der Deutschen Pathologischen Gesellschaft*, 1908, xi, 143.

Pathology.—Various types of acute cholecystitis may be differentiated—catarrhal, suppurative, phlegmonous, gangrenous, and membranous. Properly interpreted these are varying manifestations of the one process (an infection) which result from variations in the virulence of the infecting agent, in the local and general resistance and in the freedom of the circulation of the bile; they differ in degree rather than in nature, and pass sometimes almost imperceptibly the one into the others. At present we are unable to connect any of the well-known etiological agents with special types of the disease; perhaps, however, we are warranted in saying that the milder catarrhal lesions are most commonly due to the typhoid and the colon bacillus, and the suppurative lesions to the pyogenic cocci; but some of the most acute and severe lesions are provoked by the typhoid bacillus, and some of the acute rapidly phlegmonous forms are due to pyococcic infections. At present, therefore, we are forced to the conclusion that varying lesions are due not so much to different microorganisms as to variations in the virulence of the same organism and other attendant conditions.

In the mild grade of inflammation, so-called *acute catarrhal cholecystitis*, the gall-bladder is distended and tense; the wall is swollen, oedematous, and softened, and the mucosa is, in addition, congested and covered with a layer of mucus, perhaps bile-stained; in many cases the inflammatory phenomena invade also the peritoneal covering, which then is dull, lustreless, or opaque, and covered with a thin layer of fibrin which may serve to unite the gall-bladder to adjacent structures. The cystic duct usually is partially obstructed from swelling of the mucosa; sometimes it is completely obstructed, but usually only in the event of recurrent attacks of inflammation or of cicatrization of ulcerations. The contents of the gall-bladder usually consist of turbid bile; usually this is in excess and much thickened, inspissated, and tar-like; sometimes the contents consist of turbid, bile-stained, serous, or serofibrinous fluid; rarely it may be stained with blood, in which event there is likely to be some erosion or superficial ulceration of the mucosa. Gallstones may be encountered, especially in recurrent attacks; usually not in cases of less than four to six months' duration (during most of which time the infection may be latent). The adjacent lymphatic glands, especially those in the portal fissure, are usually enlarged and softened.

In the more severe, or *suppurative cholecystitis*, or acute empyema, the gall-bladder varies in size depending upon antecedent conditions. Should it have been previously normal or only slightly diseased and non-adherent, it may become considerably, sometimes very much, enlarged; but if previously the seat of fibrous cicatrization from chronic inflammation, no enlargement whatever may occur; in this case it is usually united to adjacent tissues and organs by firm adhesions. The wall of the gall-bladder is softened, swollen, oedematous, congested, and usually very dark reddish, greenish, or blackish in color. The mucosa is congested and desquamated, and covered with a fibrinopurulent, sometimes also hemorrhagic, exudation. In many cases there is more or less ulceration, especially toward the fundus, in consequence of the relatively poorer vascular supply of the fundus and the gravitation of gallstones. The ulceration may proceed through the wall and lead to perforation, with the subsequent development of a local abscess or a more general peritonitis. The cystic duct is usually occluded even in the absence of gallstones. The contents consist of turbid, bile-stained, fibrinopurulent,

sometimes sanguinolent fluid; gallstones are present in about 80 per cent. of the cases.

In some cases the phenomena are less acute; the congestion and softening of the wall are less marked or entirely absent, the wall then being thickened and indurated; there is little if any pericholecystitic fibrinous exudation, although the gall-bladder may be firmly united to adjacent organs; the mucosa of the gall-bladder, however, is more or less ulcerated and the contents are more or less purulent—cases of so-called *chronic empyema of the gall-bladder* or *chronic ulcerative cholecystitis*. On the other hand, rarely the lesions may be more acute than in the more common cases of acute suppurative cholecystitis; the œdematous, hemorrhagic, and purulent infiltration of the gall-bladder is widespread, and may lead to extensive dissection of the different coats, the separation, for instance, of the mucosa from the underlying coats, or extensive sloughing with or without perforation—so-called *phlegmonous cholecystitis*. A further or more advanced and rare stage of this process is spoken of as *gangrenous cholecystitis*, a term very inconsiderately employed. The small foci of necrosis that occur in suppurative cholecystitis, for instance, are sometimes spoken of as localized gangrene—ulcerative cholecystitis being a more preferable term. When, however, a large section of the gall-bladder becomes necrotic the term gangrenous cholecystitis is not inaptly applied. This results from very virulent infection, or interference with the blood supply, due to a gallstone impacted in the neck of the gall-bladder or in the cystic duct, or to infectious thrombosis of the nutrient artery. The lesions resemble those of advanced suppurative or phlegmonous cholecystitis, with the addition of complete necrosis or gangrene of a variable portion of the gall-bladder; the gangrene usually begins at or near the fundus (where the blood supply is poorest) and spreads toward the neck; in some cases it begins about a gallstone more or less firmly embedded in the wall of the gall-bladder. *Membranous cholecystitis* is a rare condition characterized by the formation of a membranous or fibrinous coat of the interior of the gall-bladder, and perhaps of the biliary ducts. It is usually associated with gallstones, and with mucous enteritis or colitis.

Microscopically the lesions of acute cholecystitis are those of acute inflammation in its various stages and aspects. In the mild catarrhal cases the lesions may be limited to the mucosa and the submucosa, and consist of vascular dilatation, œdema, slight cellular exudation, increased production of mucus, and desquamation of the surface epithelium; the serous coat also may reveal injection of the bloodvessels and a small amount of fibrinous exudation. In more severe cases (suppurative or ulcerative cholecystitis) the cellular exudation is more marked and necrosis and ulceration of the mucosa and submucosa occur; or there may be extensive purulent infiltration of the coats of the gall-bladder and more or less extensive sloughing (phlegmonous cholecystitis). More widespread and complete necrosis characterizes gangrenous cholecystitis.

Symptoms.—Acute cholecystitis is extremely variable in its symptomatology. In many cases it is altogether latent clinically; in other cases, although it occasions obvious symptoms, the true nature of these is often masked, and they escape correct interpretation, being commonly regarded as evidences of disordered stomach. Thus for instance during typhoid fever, but also in other circumstances, there are: (1) Cases in which a noteworthy

enlargement of the gall-bladder occurs; but the biliary ducts being patent and the drainage therefore sufficient, and the patient's sensibilities somewhat obtunded, there is no complaint and the disorder escapes clinical recognition, unless perchance systematic and repeated examinations of the gall-bladder region are undertaken; in this event a more or less enlarged and tender gall-bladder may be encountered. It is surprising how frequently this condition occurs and how frequently it goes undetected. (2) There are cases in which a little epigastric discomfort, perhaps slight nausea, in some cases actual pain, is complained of, and examination reveals an enlarged and tender gall-bladder. (3) There are cases in which, announced by the ordinary symptoms, acute cholecystitis or cholangitis develops; this is probably more common during or after convalescence than during the course of the general typhoid or other infection.

Initial nausea and vomiting, fever, pain, and tenderness in the region of the gall-bladder, rigidity of the overlying abdominal muscles, and an enlarged and tender gall-bladder constitute the important clinical manifestations of acute cholecystitis. These may be of sudden or rather insidious onset. The nausea and vomiting are usually moderate in grade and soon subside, except in the event of pericholecystitic or more general peritonitis and concomitant paresis of the intestine. In catarrhal cholecystitis the fever is slight and lasts only a short time; in the cases complicating some general infection there may be little if any added fever, but if occurring, for instance, during convalescence from typhoid fever, the onset of the cholecystitis is usually announced by fever, which may be high. The pain usually occurs first in the epigastrium, sometimes directly in the midline, and later becomes more localized to the right hypochondrium; it varies much in character. In some cases it is dull and aching; in other cases it is sharper and more severe, and while continuous is subject to acute exacerbations; in perhaps the majority it is severe and paroxysmal and resembles or is identical with the pain of so-called gallstone colic. The complaint of pain is much influenced by the mental state of the patient: in acute infections attended by mental torpor due to toxæmia there may be little or no complaint of pain, but there may be considerable local tenderness. The pain may be referred to the region of the right scapula, as in gallstone colic, or to the right shoulder when the peritoneum is involved, or downward to the right lower abdominal quadrant, and suggest appendicitis; as a matter of fact, the two conditions cholecystitis and appendicitis may be concurrent.

There is more or less tenderness in the right upper abdominal quadrant. At first this may be diffuse, but usually it soon becomes localized to the region of the gall-bladder. The overlying muscles are rigid, and in consequence sometimes simulate a tumor of the gall-bladder. In most cases, however, by careful palpation one can feel an enlarged and tender gall-bladder; in many cases it is also visible. The degree of enlargement varies considerably in different cases; in recent cases in which the gall-bladder is non-adherent and there is little pericholecystitic exudation, the tumor, corresponding with the enlargement of the gall-bladder, is pear-shaped, tense or semifluctuating, moves with respiration, and is displaceable by palpation, although both of these may be inhibited by the attendant pain; in older or recurrent cases, in cases in which there are old adhesions, and in cases in which there is considerable pericholecystitic serofibrinous and other exudation, and perhaps also involvement of adjacent tissues (with intestinal paralysis,

tympanites, abscess formation, etc.), the tumor mass is likely to be more globular or irregular, or ill-defined, and it may not move at all on respiration or be displaceable on palpation. Usually the mass is in the region of the gall-bladder, and as it increases in size it advances toward the umbilicus; but in a considerable number of cases it descends in the flank; sometimes it is found so low as to suggest peri-appendicular suppuration. With the progress of the disease the tumor often can be observed gradually to increase in size; as the inflammatory phenomena subside and the cystic duct again becomes patulous, the tumor decreases in size and may ultimately disappear. Some local tenderness especially on deep palpation, however, usually remains for some days or weeks.

Jaundice is not a part of uncomplicated cholecystitis; its development signifies extension of the inflammation of the ducts (cholangitis), spasm of the ducts, or some complication such as gallstones obstructing or compressing the hepatic or the common duct. The liver is not enlarged unless there is cholangitis. The pulse may be normal or slightly accelerated. Usually there is a moderate polynuclear leukocytosis (12,000 to 15,000).

The *consequences* of acute cholecystitis are many and various, and of much clinical importance. In the majority of cases, especially in those occurring during general infections, such as typhoid fever, the clinical manifestations subside at the end of four or five to ten or fourteen days; perhaps in some of these cases the lesions also subside and the gall-bladder returns to its previously normal condition. In the great majority of cases in which the clinical and anatomical phenomena subside the lesions become chronic, the infection becomes for a time at least latent and of minor grade: the usual consequence of this is gallstone formation; but chronic cholecystitis may ensue without gallstone formation. In many cases after the formation of the gallstones the infection gradually dies out, and the gallstones may remain latent, perhaps throughout life, although the gall-bladder in such circumstances is disposed to ready re-infection. Sometimes, especially if the infection was slight in grade and soon died out, and if a gallstone obstructed the cystic duct or the exit of the inflammatory exudation was otherwise prevented, the contents of the gall-bladder soon take on a serous or mucous character, and a condition of cystic distention or hydrops of the gall-bladder results. In other cases the gall-bladder remains continuously infected; in other cases the patient may suffer more or less continuously from minor phenomena due to chronic cholecystitis (with or without gallstones), and in addition become subject to repeated recurring attacks of acute cholecystitis (re-infections or exacerbations of a chronic infection); in either case the lesions are likely to become suppurative or ulcerative—so-called chronic empyema of the gall-bladder. Again, although the lesions of the gall-bladder subside, adhesions have developed between the gall-bladder and adjacent organs, which often engender serious consequences. In some cases of acute cholecystitis the lesions do not subside; they progress to the more serious forms of the disease—suppurative, phlegmonous, or gangrenous cholecystitis; but suppurative phlegmonous and gangrenous cholecystitis may also develop suddenly, as a primary manifestation of an infection; in about 80 per cent. of the cases, however, they occur in connection with gallstones, therefore in chronic infections or re-infections. The clinical manifestations consist of an aggravation of the symptoms of acute cholecystitis. The evidences of local inflammation, peri-

tonitis, are more severe; in some cases persistent nausea, vomiting, widespread abdominal pain and tenderness, tympanites, constipation, etc., suggest infection of the general peritoneal cavity. Occasionally the manifestations are those of acute rapidly developing general peritonitis without localizing symptoms. The gall-bladder, as a rule, is palpable—near the tip of the ninth costal cartilage in case it is incapable of little if any distention, or near the umbilicus, in the flank, or in the right inguinal region in case it has become much distended. In many cases it is quite low, being associated with cholelithiasis and linguiform lobulation (Riedel's lobe), and may simulate appendicitis. Jaundice is not a necessary symptom, but it occurs more frequently in the severer than in the milder form of acute cholecystitis, because of the associated lesions, such as gallstones and tumors obstructing the ducts, and the more common occurrence of cholangitis. The temperature is usually high (103° to 104°), and commonly accompanied by chills and sweats; but in some cases of severe infection, with minor resistance on the part of the patient, the temperature may be lower. The pulse is accelerated, 120 or more—which is a better indication of the seriousness of the patient's condition than the temperature. Usually there is a polynuclear leukocytosis of from 15,000 to 35,000 or more.

Perforation of the gall-bladder may occur: into preformed adhesions, and lead to the development of a localized abscess, which, if the adhesions involve the abdominal wall, may become manifest by local œdema and other signs of inflammation; into the liver, and lead to a liver abscess; into the general peritoneum, and lead to widespread purulent peritonitis; into the hollow abdominal viscera, and lead to fistulæ, etc. Sometimes the onset of the perforation is announced by sudden increase of the local pain, more or less collapse, increased pulse rate, and progression of the local abdominal and the general symptoms; but, as in appendicitis, the symptoms often only steadily increase, and no time can be set as that of the occurrence of perforation.

Diagnosis.—The diagnosis of acute cholecystitis presents no serious difficulties in the great majority of cases: nausea and vomiting, pain and tenderness in the region of the gall-bladder, rigidity of the overlying abdominal muscles, and an enlarged and tender gall-bladder constitute a complex of symptoms that can scarcely be attributed to anything else. In some cases the symptoms are not obtrusive, and the enlarged and tender gall-bladder must be sought, but even these cases present no diagnostic difficulties. The phenomena of gallstone colic also are properly attributable to an acute, perhaps acute exacerbation of a chronic, cholecystitis, and the local pain and tenderness and rigidity of the overlying muscles, if sought, will usually be found. The more serious forms of cholecystitis are to be distinguished from the milder or catarrhal form, usually by their more abrupt onset, or abrupt increase in severity, and by the more marked local and general symptoms, that is, more evident and perhaps widespread peritonitis and more serious toxæmia, and by the fact that in most cases there is a history of antecedent cholecystitis or cholelithiasis. The onset of perforation or gangrene can rarely be diagnosed with certainty, except perhaps by a sudden increase in abdominal pain, with collapse, increased pulse rate, and spreading peritonitis; it should be suspected in the event of sudden fulminating peritonitis occurring in a chronic cholecystitic or cholelithitic subject, especially if the lesions began in or remain limited to the upper abdomen. In this event one must exclude other causes of peritonitis of the upper abdomen,

such as perforated gastric or duodenal ulcer, pancreatitis, etc. A study of the patient's past history is of the utmost importance in this connection.

Difficulty may be experienced in differentiating acute appendicitis, particularly in those cases in which the enlarged gall-bladder is unusually low and perhaps projects into or is situated in the right ilica fossa; but cholecystitis is suggested by previous attacks of so-called dyspepsia or of more obtrusive symptoms of cholelithiasis or cholecystitis, especially if associated with jaundice, by relationship of the attacks to dietetic indiscretions, by limitation of the pain to the epigastrium or the right hypochondrium and radiation to the right shoulder (the pain is rarely as diffuse as is often in early appendicitis), by the presence of a zone of cutaneous hyperalgesia (Head) about the upper right half of the abdomen, by the fact that the mass (enlarged gall-bladder) often moves with the liver during the phases of respiration, etc.

Prognosis.—In the majority of cases the prognosis in acute cholecystitis is good, the process tending to subside, under appropriate treatment, within ten days or a fortnight; the mildest cases, of which doubtless many are overlooked, may subside within four or five days or a week; the inflammatory and retained secretions are discharged through the cystic duct and the gall-bladder reverts to its normal size. Sequels, especially gallstones and adhesions, however, are almost certain to occur, so that the subsequent outlook is not the best; in other cases a low-grade infection may persist and the patient become a subject of chronic infection and recurring attacks of cholecystitis. In the more severe forms, suppurative, phlegmonous, and gangrenous cholecystitis, the tendency toward spontaneous cure is slight, in this event the prognosis is grave, since not only are the lesions of the gall-bladder serious in themselves, but a localized abscess or general peritonitis may ensue and a fatal issue result, unless recourse is had to surgical intervention. Occasionally in these serious cases the obstruction of the cystic duct may be due only to swelling of the mucosa; in this event it may lessen and part at least of the contents of the gall-bladder may be discharged; but if the acute inflammatory lesions subside, chronic empyema is likely to ensue. In some cases the infection is so virulent that extensive phlegmonous inflammation or gangrene develops within a few days, before limiting adhesions can form; the prognosis in these cases is extremely grave, death usually resulting unless the patient is operated upon.

Treatment.—The treatment in general is that recommended for gall-stone colic, which virtually is an attack of more or less acute cholecystitis. The diet should be reduced to a minimum; perhaps withheld altogether for a day or two, if the patient's general condition permits. Until the acute phenomena subside the diet should be bland and easily digested: milk and Vichy or lime water, broth, cereals, etc. The bowels should be opened by enemas of hot water for the first day or two, whereupon Carlsbad salts or sodium phosphate or similar salts in hot water should be given; the enemas of hot water may be continued, since they serve to deplete congestion of the intestinal and biliary tracts. In the absence of severe pain it is wise to withhold morphine, which, unless required to mitigate acute suffering, tends to mask symptoms, to promote a false security, and is otherwise objectionable. Relief of the pain usually follows hot fomentations to the region of the bladder, lavage of the stomach with hot alkaline water, a hot full bath, etc.; if not, a small dose of morphine ($\frac{1}{16}$ to $\frac{1}{32}$ grain) may be tried;

a very small dose is often extremely efficacious. Nausea and vomiting depend upon the same factors as the pain, and they usually subside with the pain; should they persist, they may be controlled by bismuth, carbolic acid, creosote, diluted hydrocyanic acid, cerium oxalate, and sips of hot water, brandy, champagne, etc. Following the subsidence of the acute phenomena the treatment recommended for cholelithiasis should be instituted.

Happily most cases of acute cholecystitis gradually subside; occasionally, however, the local symptoms increase, evidences of more or less extensive peritoneal irritation or actual peritonitis supervene, the temperature, leucocytosis, and other manifestations of infection and toxæmia increase: the local lesions have progressed to suppuration, necrosis, or gangrene. In this event the necessity of operative intervention must be seriously considered. It is wise, if possible, to let the acute manifestations subside without surgical assistance, but when they are obviously increasing, operation should not be delayed.

CHRONIC CHOLECYSTITIS.

Etiology.—Chronic cholecystitis may be the residual manifestation of an acute cholecystitis, or the process may be chronic from the beginning. Its etiology is that of acute cholecystitis, the chronicity of the process being a manifestation of lingering infection (which is common), or, the consequence of very low-grade infection with almost but not quite sufficient biliary drainage. In the great majority of cases the condition is associated with gallstones, the one process sustaining the other; that is, most cases are calculous cholecystitis; in a few cases the lesions extend throughout the biliary tract; that is, cholangitis and cholecystitis are associated, the one or the other being in the ascendancy.

Pathology.—As stated, the process may or may not be associated with gallstones. In the absence of gallstones the lesions may be catarrhal, that is virtually confined to the mucosa, which shows more or less congestion, swelling, desquamation, and increased formation of mucus; the gall-bladder usually is distended and filled with thick tenacious bile or bile and mucus, which may contain typhoid bacilli, colon bacilli, etc. In some cases the mucosa is thinned and atrophic, especially in spots, evidently the consequence of past erosion or ulceration. In many cases of long standing the gall-bladder becomes reduced in size, very small, and sclerotic; it may be represented by a mass of thickened contracted connective tissue, perhaps tightly enclosing one or more gallstones, but sometimes not. When thus small and shrivelled, the gall-bladder, whether containing gallstones or not, often makes up part of a mass of firm adhesions that bind together the adjacent tissues; in many cases the gall-bladder can scarcely be identified as such. Cholesterin deposits and calcareous infiltration are common. The cystic duct is often, perhaps usually, patulous, but it may be partially or completely obstructed. In some cases the tissue adjacent to the gallstones, or, in their absence, that lining the still-persisting lumen, is made up largely of cicatrizing granulation tissue, and may reveal no vestige of mucosa; in other cases the lining epithelium has invaded the deeper layers of the walls and is clearly hyperplastic and irregularly proliferated (beginning carcinoma). The musculature is usually atrophic, and replaced by fibrous connective tissue.

Symptoms.—The symptoms are those of cholelithiasis—which is the important concomitant of chronic cholecystitis. A clinical differentiation between cholecystitis with and without cholelithiasis may perhaps be attempted, but those with most experience find it increasingly difficult to determine from the clinical signs whether or not gallstones are present in individual cases.

Diagnosis.—It is practically impossible in most cases to say definitely whether or not gallstones are present. The likelihood of their being present is enhanced by repeated attacks of severe colic followed by jaundice, but colicky pains are present in many cases (70.6 per cent.) without gallstones, and jaundice may occur in 35.3 per cent. of cases without gallstones. The detection in the stools of faceted gallstones suggests the presence of others in the gall-bladder. The absence of gallstones in a case of chronic cholecystitis can scarcely be postulated with the knowledge at hand.

The *prognosis* and *treatment* are the same as in cholelithiasis.

CHOLELITHIASIS.¹

Etiology.—Cholelithiasis is an exceedingly common condition, being found at the necropsy in from 5 to 10 per cent. of subjects dead from all causes. It is especially prevalent in the temperate zones, being quite uncommon in the tropics. It occurs at all ages, but the incidence increases progressively with advancing years: 75 per cent. or more of the cases are found in persons over forty years of age, and less than 1 per cent. in those under twenty years; but this age incidence does not correctly represent the time of the formation of gallstones, since in many cases, having formed, they remain latent for years and are found only when the subject dies at a more advanced age from the consequences of gallstones or other causes. Doubtless, therefore, many if not most gallstones are formed in early adult life, before the fortieth year, corresponding with the period of greatest frequency of typhoid and other infections, etc. Rarely the disorder is encountered in infancy or childhood. The majority of cases found in infancy are doubtless due to intra-uterine infection, the process perhaps being analogous to that which in other circumstances induces congenital obliteration of the bile ducts; the cases found in older children also may have originated during foetal life and remained latent for a number of years, but it is not always possible to exclude postnatal typhoid and other infections. Gallstones are

¹ The literature of cholelithiasis is extensive and is being continuously added to in the current periodical literature. The more important monographs comprise: Naunyn, *A Treatise on Cholelithiasis*, 1896; Kehr, *Gallstone Disease*, 1901; Mayo Robson, *Diseases of the Gall-bladder and Bile Ducts*, third edition, 1904; Moynihan, *Gallstones and their Surgical Treatment*, second edition, 1905; Bland-Sutton, *Gallstones and Diseases of the Bile Ducts*, 1907. Virtual monographs are contained also in Nothnagel's *Encyclopedia of Practical Medicine*, article by Hoppe-Seyler, and in Rolleston's *Diseases of the Liver, Gall-bladder, and Bile Ducts* (literature). Valuable articles by Ewald, Musser, Brewer, Herter, Mayo, and Kehr are contained in the *Transactions of the Congress of American Physicians and Surgeons*, 1903, vi. The results of considerable personal experience will be found in the following articles: Deaver, *American Journal of the Medical Sciences*, 1908, cxxxv, 137, cxxxvi, 625; Mayo, *Annals of Surgery*, 1906, xlv, 209; Richardson, *Boston Medical and Surgical Journal*, 1906, clv, 329, 1907, clvi, 687; and Kehr, *Deuxième Congrès de la Société Internationale de Chirurgie*, Bruxelles, September 21–25, 1908.

more common in women than in men, the ratio being variously stated as 5 to 1 (Schroeder) to 4 to 3 (Rolleston).

The older writers attributed much influence to heredity, but in later days this is less believed in. Evidence in favor of so-called "arthritic" and other diatheses grows progressively less, and assuredly the essential factor in the formation of gallstones, the infection, cannot be inherited; but many contributing factors are common to many members of the same family, such as sedentary habits, lack of exercise, obesity, dietetic indiscretion, etc., which doubtless are of more etiological significance than an inherited family disposition.

The one necessary factor is a low-grade catarrhal inflammation of the biliary tract; the second requisite seems to be some obstruction to the free flow of bile. The one without the other factor is not sufficient; whether a third factor is of significance remains to be determined. The catarrhal inflammation of the biliary tract is set up especially by typhoid and colon bacilli; but also apparently by attenuated streptococci, staphylococci, and other microorganisms that may cause acute cholecystitis; perhaps also by anaërobic bacteria.¹ It is essential that the antecedent catarrh be mild in grade, since in severe infections the mucosa of the gall-bladder is likely to be more or less destroyed and its cholesterin-producing function abolished. The infective agents usually reach the gall-bladder by way of the portal circulation; variations in the source and pathways of infection are similar to those already mentioned in connection with acute cholecystitis.

Apart from infection of the biliary tract, a secondary requisite seems to be some obstruction to the free flow of bile. This occurs in circumstances similar to those mentioned in connection with acute cholecystitis; but there are also a number of additional contributing factors. Thus gallstones are especially common in those who lead a sedentary life as contrasted with laborers and others who work much outdoors, in women as contrasted with men, etc.; as part of the general muscular inactivity, the abdominal muscles and the diaphragm, contract relatively feebly and the bile, inefficiently expelled, stagnates in the gall-bladder. Similar consequences ensue upon obesity and disorders which interfere with the free movement of the diaphragm.

In women a number of factors contribute: in addition to a more sedentary life, they are more often the subject of hepatoptosis or nephroptosis, brought about by repeated pregnancies and other factors, that occasion more or less marked and continuous distention of the abdomen and interfere with the movements of the diaphragm. In consequence of the prolapse of the liver, the gall-bladder becomes dependent and the cystic or the common bile duct kinked, or perhaps has considerable traction brought to bear upon it and becomes obstructed, so that the gall-bladder is less easily emptied than in health and is more disposed to infection. Tight lacing may act in a similar manner. The frequency of pelvic infections may be of some etiological significance, either by serving as the source of the infection or leading to the formation of adhesions. The association of cholelithiasis with pregnancy is undeniable, but its importance is difficult to estimate, since the great majority of middle-aged women, whether or not they suffer from gallstones,

¹ Consult Gilbert and Lippman, *Compte-rendus de la Société de biologie*, 1907, lxiii, 405.

have been pregnant; there is some evidence, however, that gallstones are more common in those who have been pregnant, especially repeatedly pregnant, than in those who were never pregnant. Perhaps in some cases puerperal infections are the cause of the gallstones; sometimes the biliary infection, although often misinterpreted, can be definitely determined to have been acquired during the puerperium. Cholelithiasis is sometimes found in association with appendicitis; when the appendicitis precedes, the cholelithiasis and cholecystitis may have resulted from the transport of infection by the portal circulation; when the cholecystitis precedes, the appendicitis may perhaps result from the lodgement of infection in an unusually vulnerable organ. Colwell¹ believes that gallstones are more common in carcinomatous than in non-carcinomatous subjects.

Gallstones show a relatively increased incidence in certain forms of heart disease, especially mitral and aortic valvular disease. This is doubtless due to several factors: the more sedentary mode of life incumbent upon chronic cardiac subjects, and the chronic congestive processes which obstruct the flow and perhaps cause inspissation of the bile, reduce the resistance of the biliary tract, and favor infection. Similar factors account for the relative increase in the incidence of gallstones in certain chronic pulmonary diseases.

Diet, disorders of the gastro-intestinal tract, and disorders of metabolism have long been credited with more or less etiological significance, but their exact influence is difficult to determine. Disorders of the gastro-intestinal tract associated with catarrh doubtless are of significance, since they may lead to extension of congestive processes to the biliary tract, provide bacteria that may induce catarrhal disorders in the bile ducts, and are a fruitful source of many toxic fermentative and decomposition products that also exert a deleterious influence on the biliary passages. Dietetic and other indiscretions that lead to gastro-intestinal catarrh, therefore, are of at least indirect influence in causing gallstones; and a vicious circle is formed in that cholelithiasis and the antecedent cholecystitis not only are frequently manifested by misinterpreted symptoms of so-called dyspepsia or indigestion, but lead directly to gastro-intestinal catarrh, and in virtue of the commonly associated precholecystitic adhesions induce much more serious disturbances of the stomach and intestines. Constipation, an expression of or associated with dietetic indiscretions, sedentary habits, etc., is probably of some etiological significance in itself, largely on account of the attendant sluggishness of the intestinal circulation—which favors congestion and infection of the biliary tract.

Whether diet as such is of real etiological significance is difficult to determine. Heavy eating and the excessive use of alcohol are doubtless of much importance, especially by virtue of the gastro-intestinal catarrh and the chronic congestion of the portal system that they lead to; whether they are of additional significance, by virtue of disturbances of metabolism, is not definitely known, although it is widely and has long been believed in. Perhaps an excessive amount of food is of more significance than its character, although it is said that a carbohydrate or fatty diet disposes to, and a proteid diet disposes against, the formation of gallstones. The matter, however, is largely speculation; since it has been definitely determined that the precipitation of

¹ *Archives of the Middlesex Hospital*, 1905, v, 142.

cholesterin is due to catarrh of the biliary tract, the supposition that a carbohydrate diet leads to such precipitation by reducing the bile acids, the solvents of cholesterin, has lost much of its supposed importance, and this has been totally destroyed by Naunyn's statement that even in diseased conditions the bile scarcely if ever contains so much cholesterin that it cannot take more in solution. The relative unimportance of diet is further suggested by the fact that although German soldiers are said to suffer from cholelithiasis twice as much as English soldiers, which has been attributed to the fact that the German has a ration of only one-half as much meat as the English soldier, the native Indian soldier, who subsists on an almost exclusive carbohydrate diet, suffers scarcely if at all from cholelithiasis. The presumed comparative freedom from cholelithiasis of diabetic subjects, which has been attributed to an excessive proteid diet, is probably a misinterpretation of surface phenomena, and is somewhat negated by the frequency of gallstones in gouty subjects who also consume considerable proteids. Perhaps restriction in the amount of fluid intake may be of some significance by causing inspissation of the bile, but this is rarely if ever operative.

The matter has been well expressed by Herter,¹ who says: "It is plain from what has been said that there is at present no unequivocal evidence that gallstones arise from constitutional derangements unconnected with micro-organismal invasions of the gall-bladder. On the other hand, it is certain that the cholesterin of the bile can be considerably increased by local irritants unconnected with infection, and it is likely that the requisite local conditions for such increase sometimes arise through purely metabolic disorders. While gallstones are commonly the result of local infections, we should carefully guard against the conclusion that they can never have a diathetic origin. It is at least highly probable that diathetic conditions are capable of so altering the composition of the bile as to favor materially the production of calculi in the presence of suitable local bacterial activities." Beer² also believes that a third factor, which he says may be called altered liver metabolism or a diathesis, may be of significance in the etiology of gallstones. He believes that Naunyn's factors, stagnation of bile and inflammation of the biliary passages, do not seem to be sufficient in themselves to lead to gallstone formation, even though the time allowed for the working of the causes be adequate, and that these factors lead to gallstones only in persons who previously have had gallstones.

Pathology.—Gallstones may be single or multiple; that is, there may be one or there may be thousands (7802 in a case of Otto's). In perhaps most cases the number varies between a dozen and a hundred; most of those in excess of a hundred are small and inconsequential, and are spoken of as biliary sand or gravel. The stones vary much in size—from the smallest particle to those larger than a normal gall-bladder. Merkel found a gallstone measuring 15 cm. in length and 12 cm. in circumference; and Frerichs reports one that weighed 120 grams, and Ritter one that weighed 135 grams. When there are many stones, most of them are usually small; when there are few, there may be several rather large stones; when there is only one, it may be small, moderate in size, or very large, even completely filling a distended gall-bladder. Gallstones vary also in shape. The

¹ *Transactions of the Congress of American Physicians and Surgeons*, 1903, vi, 158.

² *American Journal of the Medical Sciences*, 1905, cxxx, 432.

large single stones, or several stones that fit together (obviously resulting from fracture of a single large stone), conform to the shape of the interior of the gall-bladder, and are therefore ovoid or somewhat pear-shaped. Small single stones are usually ovoid or spheroidal. Multiple stones are usually cuboid or polyhedral, and exhibit variously shaped surfaces (facets), triangular, quadrangular, polygonal, etc., which being opposed to one another are usually smooth and polished, although it is doubtful if this results, as is supposed, in consequence of mutual attrition; the edges of these stones are rounded rather than sharp. Stones within the intra-hepatic ducts conform to the ducts in shape, and are ovoid or cylindrical; occasionally they are branched, as are the ducts. Although usually smooth, the surface of gallstones may be rough, irregular, or nodular (mulberry calculus). In consistency gallstones vary, depending upon the age and the chemical composition, the recently formed and the cholesterol stones being the softest; older stones may be quite hard, but sometimes they crumble readily, especially after being dried.

The following varieties of gallstones are distinguished by Naunyn: (1) Pure cholesterol stones, which consist of nearly pure cholesterol and are uncommon. They vary in size from that of a cherry to that of a pigeon's egg, are hard, oval or roughly spherical, seldom faceted, have a smooth or nodular surface, and are pure white or yellowish and translucent, or rarely brown, greenish, or brownish black on the surface. On section they are white and crystalline throughout, or show brown deposits between the crystals; they are not stratified. (2) Laminated cholesterol stones, which consist of about 90 per cent. of cholesterol, together with bilirubin calcium, biliverdin calcium, and calcium carbonate. In general appearance they resemble the pure cholesterol calculi, but they may be brittle and friable, and they are often faceted. On section they are more or less distinctly laminated, layers of almost pure white alternating with others that are yellow, brown, green, or red. The centre may be crystalline, but the external layers are usually vitreous or earthy. (3) The common (or mixed cholesterol) stones, which vary much in size, although they seldom equal a large cherry; they may be small and very numerous, and are usually faceted. The surface is usually yellow, but often brown or white. When fresh they are often soft and greasy; when dried they undergo shrinkage and become harder. On section the centre is often soft and may contain a cavity. (4) Mixed bilirubin-calcium stones, which may consist roughly of 75 per cent. of bilirubin calcium, and about 25 per cent. of cholesterol. They are as large as a cherry or larger, and may occur singly or in groups of three or four in the gall-bladder or larger ducts. Their shape depends upon their situation; when multiple they may be faceted. They consist of concentric layers of reddish-brown or dark-brown material, which is seldom quite hard, and contracts on drying often with the formation of fissures or cracks. (5) Pure bilirubin-calcium stones, which vary in size from that of a grain of sand to that of a pea. There are two types: small, solid, brownish-black concretions, with rough irregular surfaces and wax-like consistency, and exhibiting a disposition to become welded; and harder, smooth concretions of a grayish-black metallic lustre and an internal spongy structure. (6) Rarer stones, such as amorphous and incompletely crystalline cholesterol gravel (resembling pearls); calcareous stones; concretions with included bodies and conglomerate stones; and casts of the bile ducts.

Gallstones may be found in any part of the biliary tract. Of 216 patients operated upon for infections of the biliary tract by Dr. John B. Deaver, at the German Hospital, Philadelphia, 182 (84.2 per cent.) had gallstones and 34 (15.8 per cent.) had no gallstones. Of the 182 that had gallstones, 101 (55.5 per cent.) had stones in the gall-bladder alone; 23 (12.5 per cent.) had stones in the gall-bladder and cystic duct; 19 (10.5 per cent.) had stones in the gall-bladder and common duct; 12 (6.6 per cent.) had stones in the common duct alone; 11 (6.0 per cent.) had stones in the cystic duct alone; 5 (2.8 per cent.) had stones in the gall-bladder, cystic, hepatic, and common ducts; 4 (2.2 per cent.) had stones in the gall-bladder, hepatic, and common ducts; 1 (0.6 per cent.) had stones in the gall-bladder, cystic, and common ducts; 2 (1.1 per cent.) had stones in the adhesions (not otherwise specified); and 4 (2.2 per cent.) had stones in regions not definitely specified. Of the 216 patients, 58 had no stones in the gall-bladder, and of these, 23 had stones in the ducts; 41 had stones in the common duct, and of these, 12 had stones nowhere else; 9 had stones in the hepatic ducts (as well as elsewhere).

Pathogenesis.—In consequence of bacterial infection of the biliary tract a low-grade catarrhal inflammation is set up; this, on the one hand, leads to obstruction to the free flow of bile from swelling of the mucous membrane, and the products of this inflammation, on the other hand, contain the essential constituents of gallstones. In the gall-bladder the catarrhal inflammation leads to desquamation of the lining epithelium, an albuminous exudation, and an increased formation of mucus and of cholesterol; and in the gall-bladder dynamic factors frequently favor the stagnation of bile. The increased cholesterol is derived not from the bile; it results from catarrhal disintegration of the mucous cells lining the wall of the gall-bladder. The second important constituent of the gallstones, bilirubin-calcium, is derived from the bile, being precipitated by bacterial growth or by the albuminous exudation of the inflammatory process—a phenomenon that can be imitated experimentally by adding egg albumin to normal bile. Recently, Bacmeister¹ and Exner and Heyrovsky² have shown that bacteria, notably the typhoid and the colon bacillus (but not streptococci), may effect directly a decomposition of the bile with the precipitation of cholesterol. Exner and Heyrovsky attribute this to decomposition of the bile-acid salts (the solvent menstruum), in consequence of which the normal amount of cholesterol can no longer be held in solution. Stagnation and inspissation of the bile do not give rise to the increased formation of cholesterol nor to the precipitation of bilirubin-calcium—whence they are contributing factors only. Bilirubin-calcium is believed by Naunyn to act as a cement substance, binding together the cholesterol, desquamated epithelium, etc., the whole forming the nucleus of the gallstone. The exact significance of foreign bodies and of chemical substances apart from bacterial products has not yet been definitely determined. There being no cholesterol-bearing mucous membrane in the smaller bile ducts, cholesterol gallstones are not formed within the intrahepatic ducts, although by a retrograde movement they may be transported thither; bilirubin-calcium calculi only are formed in the intrahepatic ducts; but both varieties, as well as mixed calculi, are formed within the gall-bladder.

¹ *Münchener medizinische Wochenschrift*, 1908, lv, 211, 283, 339.

² *Wiener klinische Wochenschrift*, 1908, xxi, 214.

Associated Lesions.—Since gallstones result from catarrhal inflammation (infection) of the biliary tract, the lesions of cholecystitis and cholangitis are common concomitants—so-called calculous cholecystitis and cholangitis. The gallstones may be free in the gall-bladder or the ducts, although usually more or less confined by the associated chronic inflammatory processes; in some cases, the stones may be quite adherent to the mucosa, especially in the gall-bladder; in other cases there is more or less incrustation of the wall, a deposition in the wall of cholesterol, a process analogous to the formation of gallstones in the lumen of the gall-bladder.

The consequences of gallstone activity may be mechanical or inflammatory or both. The mechanical consequences follow what may be designated wandering of the stone into and through the biliary passages. In consequence of this activity, the gallstone may set up mechanical effects which are of great variety and of varying importance. Probably the most important and serious comprise permanent complete obstruction of the common bile duct, with consequent permanent jaundice, dilatation of the bile ducts and so-called biliary cirrhosis of the liver. Of only less importance are the dragging sensations and discomfort attendant upon the weight of many gallstones in a distended gall-bladder; the gradual production of a Riedel's or a linguiform lobe of the liver; the effects of pressure on adjacent organs; complete obstruction of the cystic duct, which, as a rule, leads at first to some distention of the gall-bladder, but soon to absorption of the bile and its replacement by mucus (hydrops) and gradual shrinkage of the gall-bladder, etc. These, however, acquire their major importance from the opportunity that they afford for bacterial infection, which is usually a concomitant condition.

This infection of the biliary tract is of the utmost significance, and forms an integral part of so-called calculous cholecystitis and cholangitis. The ensuing lesions are of the greatest diversity. The concomitant inflammatory phenomena may be of varying grades—from the mildest catarrhal lesions to widespread phlegmonous and ulcerative processes; gallstones, if present, may be quiescent or active; they may cause an acute or chronic, partial or complete, temporary or permanent, obstruction of the cystic, the hepatic, or the common bile ducts, and, on the other hand, such obstruction may occur in the absence of gallstones (being due to swelling of the mucous membrane, kinking of the ducts, or obstruction from without), and in the presence of gallstones the ducts may be partially or completely patulous; the gall-bladder may be distended or contracted, its walls thinned or much thickened, and its lumen ultimately may become almost if not quite obliterated; it may contain bile, mucus, blood, or pus, or combinations of these, in addition to or in the absence of gallstones; adhesions may form between the gall-bladder and adjacent structures, and by way of the adhesions, the gallstones may rupture into the gastro-intestinal tract and sometimes cause intestinal obstruction; or purulent pericholecystitis and pericholangitis, localized or generalized peritonitis, pylephlebitis, pericholangitic abscesses of the liver, fistulae, acute and chronic pancreatitis, etc., may ensue; and finally, in some cases a general bacterial, often pyococcic infection, with or without multiple abscesses, may develop.

Symptoms.—The symptomatology of gallstones is extremely variable, and one may speak perhaps of the immediate and the remote consequences of gallstones, but in so many cases do the one run into the other that exact clinical differentiation is often quite impossible. One may also speak of

the mechanical and the infectious phenomena, the aseptic and the septic phenomena of the French writers; this in many respects is a serviceable classification, but the one set of phenomena by no means can always be differentiated from the other, and the one frequently follows the other or they occur together. The symptomatology is further complicated by the fact that in different cases the symptoms may be due to the gallstones themselves, or to the associated infectious cholecystitis or cholangitis, pericholecystic adhesions, disease of related or adjacent organs, etc. One may, however, differentiate (1) a general symptomatology, and (2) certain special phenomena due to obstruction of the cystic duct or (3) of the common duct, and (4) certain complications and sequels.

General Symptomatology.—The characteristic and significant symptoms are: (1) Chronic, long-continued, or recurring indigestion, and (2) the phenomena designated gallstone colic. The symptoms of chronic long-continued or recurring indigestion are of the utmost importance and are commonly misinterpreted. This stands in relationship with the facts—that gallstones are present in very many subjects and are commonly believed not to cause noteworthy symptoms; that symptoms, having once occurred, are exceedingly likely to recur; and that the symptoms are often paroxysmal and very severe.

It is often said and widely believed that gallstones are usually latent, and that only a very small percentage of gallstone subjects (commonly estimated at 5 per cent.) manifest noteworthy symptoms thereof. Gallstones are sometimes latent, particularly in elderly subjects in whom the original infection has died out, and in whom more or less atrophy, especially of the muscular coat of the gall-bladder and biliary ducts, has occurred; but it is now well known to surgeons, although less widely acknowledged by general practitioners, that the symptoms commonly denominated "stomach trouble," "indigestion," "dyspepsia," etc., are due in many cases to disease of and about the biliary tract. In a number of cases in which gallstones are an accidental finding at the necropsy, investigation of the past history reveals many and often long-continued attacks of "indigestion;" the stomach is often found normal—in which event the gallstones, cholecystitis, or precholecystic adhesions, rather than being accidental, serve to explain the clinical symptoms. We must renounce the tenet that gallstones in the great majority of cases cause no symptoms: they do cause symptoms—symptoms, however, referable often to the stomach rather than to the biliary tract; that is, they may cause symptoms for years without giving rise to jaundice or gallstone colic.

Gallstone colic usually develops suddenly with severe, often agonizing, pain in the right hypochondrium or the epigastrium, radiating around the chest or to the right scapular region; nausea, vomiting, and prostration—weak, rapid pulse, rapid heart action, profuse perspiration, etc. Sometimes the attack is preceded by local, less acute pain or discomfort, chilliness, fever, etc.; more commonly (60 per cent. or more of the cases) chilliness and fever accompany and follow the pain. In some cases the severe pain lasts for a few moments only; usually it lasts for from two to twelve hours; in unusual cases it may last much longer, but in these circumstances the phenomena are more correctly interpreted as a succession of paroxysms. Often the pain ceases as suddenly as it began, but usually following the agonizing pain, more or less dull aching continues for a variable period. The sudden cessa-

tion of the severe pain may be interpreted as a sudden relief of tension to which the gall-bladder has been subjected, due to opening of the cystic duct by the dropping of an obstructing gallstone into the gall-bladder or the passage of the stone from the common duct into the intestine; in this event the calculus may subsequently be found in the stools. The stools should always be searched for calculi by softening them in water or a carbolic acid solution, and passing them through a sieve.

In the great majority if not in all cases local examination reveals tenderness, abdominal rigidity, and the other local phenomena of acute cholecystitis. The colic may recur at varying intervals—days, weeks, months, or years; occasionally there is only one attack, usually there are many; with succeeding attacks the likelihood of other attacks increases.

The pains of cholelithiasis may be colicky or non-colicky. Although there is some discussion as to the cause of the colicky pain, there is little or no objection to attributing the non-colicky pain to inflammatory phenomena provoked by infection, inflammation of and about the gall-bladder and biliary ducts. The dull aching pains are with excellent reason ascribed to inflammatory changes in the gall-bladder, with the exudation of inflammatory products and consequent distention; the more acute pains, associated with muscular rigidity, tenderness, etc., are due to infection of the regional peritoneum, which as it subsides occasions peritoneal adhesions, a contributing factor in the dull aching pains, and of other significance. Some considerable distention of the gall-bladder is possible, however, without provoking noteworthy pain, provided the cystic and the common ducts are patulous. It is to efficient drainage that the comparatively symptomless course of some cases of cholelithiasis is to be attributed, although the absence of inflammatory phenomena in the wall of the gall-bladder and the ducts, and the absence of pericholecystic adhesions, are also of significance. As regards the referred pains, although the sympathetic nerve is of significance, the demonstration that filaments of the pneumogastric nerve are distributed to the gall-bladder serves to explain the common association of gastric and cardiac symptoms with disease of the biliary tract; and the frequent occurrence of pain, sometimes of actual colic, three or four hours after a meal, suggests a causal relationship to the normal physiological contraction of the gall-bladder induced by the entrance of the chyme into the intestine.

As regards the colicky pains, there is no doubt that heretofore we have been too much concerned with interpreting them as evidence of the passage of a gallstone through the biliary ducts; pains of like character may doubtless be due to different factors, which have been well pointed out by Riedel, who gives the following as the causes of gallstone colic; (1) Adhesions of a gall-bladder no longer containing stones; (2) adhesions when large stones are present in the gall-bladder and the cystic duct is patent; (3) inflammatory processes in a gall-bladder distended by fluid or stones, when the cystic duct is occluded by inflammation or by the presence of a stone in the neck of the gall-bladder; (4) the transit of a stone through the bile passages; and (5) the inflammation of a dilated, calculous common duct, or its tributaries, without impaction of the stone.

There can be little doubt that in a number of cases the spasmodic efforts of the gall-bladder and the common duct to expel their contents by vigorous contraction of their muscular coats are the cause of the peculiar gallstone

colic. This pain occurs in its greatest intensity during the transit of a stone through the ducts, and it ceases as the stone is discharged into the intestine or drops back into the gall-bladder—thus freeing the cystic duct. But there also can be little doubt that attacks of pain indistinguishable from those provoked by the transit of a stone may be due to other factors—those mentioned above; although in some instances the attacks of pain may not be as severe as those of a so-called true gallstone colic. If the obstruction be incomplete, hypertrophy of the muscular coat results, and later often lessening of the size of the cavity; whereas, if the obstruction be complete, paresis of the muscular coat soon occurs, dilatation ensues, and the pain subsides. But, as exemplified by the 216 patients already mentioned, gallstones are absent in 15.5 per cent. of the patients who have colicky pains, and they are present in 77.7 per cent. of the patients who have only non-colicky pains.

Furthermore, as is well known, gallstones may be passed by the bowel, that is, they may ulcerate into the bowel, forming a fistula, without colicky pains having ever been experienced by the patient. As regards the cause of the colic, although mechanical factors, such as traumatism, joltings, etc., may sometimes be the immediately provoking factor, it results in the great majority of cases from infection and consequent inflammation; that is, cholecystitis provokes activity by giving rise to an inflammatory exudation that distends the gall-bladder, which, becoming tense, contracts upon its contents in an effort to expel them, and, should a stone be present, drives it into the neck of the gall-bladder and perhaps into the cystic duct.

Cases of protracted colic may be looked upon as due to long-sustained efforts of the gall-bladder to overcome the obstruction to the egress of its contents, or to slow progress of a calculus along the ducts, the consequence of the large size of the stone or the spiral arrangement of the Heisterian valve which retards progress. Gallstone colic, therefore, although it is often due to the passage of a gallstone along the ducts, should be interpreted rather as evidence of acute cholecystitis (or acute exacerbation of chronic cholecystitis): an interpretation sustained by the concomitant occurrence of enlargement and tenderness of the gall-bladder, fever, chilliness, leukocytosis, etc.—the ordinary signs of acute cholecystitis. Furthermore, as the inflammation subsides the stones become quiescent and the symptoms subside.

Jaundice is a common but not a necessary symptom of cholelithiasis, and when it occurs, may come on a variable time after the pain—a few hours or several days; that is, when the obstruction has reached the common duct. It varies in degree and duration with the local conditions. If the obstruction results from the traumatism and consequent catarrh due to the passage of a stone it may be moderate in grade and pass off within a week or thereabouts—as the catarrhal swelling subsides and the duct becomes again patulous. Should the obstruction be more complete and the stone not be discharged, the jaundice may become quite extreme and lasting; but a very large stone, as large as the distal extremity of one's thumb, may be in the common duct without causing jaundice.

The jaundice of cholelithiasis is mechanical or obstructive, and may be due to (1) gallstones in the hepatic or the common bile duct; (2) spasm of the musculature or inflammatory swelling of the mucosa of the biliary ducts; and (3) compression of the common duct or of the extrahepatic part of the hepatic duct by a large stone in the cystic duct, swollen lymph glands,

regional tumors, inflammatory exudations, adhesions, kinking of the ducts, etc. We may, therefore, speak of a lithogenous jaundice, an inflammatory (or infectious jaundice), and a compression jaundice—ill advised as these terms may be on some occasions. The practical deductions to be drawn from the known facts are: (1) That in some cases of cholelithiasis, jaundice is not due to obstruction of the ducts by a stone, but rather to infection, inflammatory swelling of the ducts, and (2) that in many cases of cholelithiasis jaundice does not occur at all, although other important and distressing symptoms may be quite obtrusive. Of 216 patients, 74 (34.2 per cent.) never had jaundice; 141 (65.3 per cent.) had no jaundice at the time of operation; whereas 121 (56 per cent.) gave a history of attacks of jaundice prior to the time of operation. These attacks, in some cases, antedated the operation many years; in some cases long periods of freedom from jaundice followed; in some cases there were no subsequent attacks of jaundice whatever.

The gall-bladder may or may not be enlarged and palpable: Thus of 216 patients, in 88 (40.7 per cent.) the gall-bladder was enlarged; in 9 (4.1 per cent.) the gall-bladder was normal in size; in 32 (15 per cent.) the gall-bladder was small and atrophic; and in 87 (40.2 per cent.) the size of the gall-bladder is not known.

Special consideration has been given to the diagnostic importance of the size of the gall-bladder since a knowledge of what is known as Courvoisier's law has become general. Courvoisier, basing his opinion upon an analysis of 187 cases, stated that in cases of chronic jaundice, contraction of the gall-bladder is suggestive of gallstones, and that dilatation of the gall-bladder is suggestive of biliary obstruction caused by factors other than gallstones—that is, pressure from without, most often carcinoma of the head of the pancreas. This so-called law serves well in the majority of cases, although as Courvoisier himself admitted, there are some notable exceptions to its universal applicability. Unfortunately, the case histories under review are not in all respects sufficiently detailed to permit a final statement in this particular; but it is of some interest to know that in at least 40.5 per cent. of 116 patients who had gallstones and jaundice, the gall-bladder was enlarged. In somewhat more than one-half of these cases, however, the gallstones were present in the gall-bladder alone, and the associated jaundice was doubtless due to inflammatory swelling, pressure from without, etc. Courvoisier's explanation of the shrinkage of the gall-bladder in these cases is correct; that is, it is due to inflammatory thickening and cicatrization, the consequence of repeated infection. This process requires time—whence the condition of the gall-bladder varies early and late in the disease; that such cicatrization does occur is suggested by its presence in a number of cases in which gallstones were present without jaundice. It is manifestly impossible that such a thickened and cicatrized gall-bladder should dilate, no matter what the obstruction in the common duct. One can conceive, however, of an acute cholecystitis or empyema of the gall-bladder (with distention) and an acute impaction of a stone in the common duct (with jaundice); or of hydrops or empyema of the gall-bladder (with distention) and a large stone in the cystic duct compressing the common duct (with jaundice); or of the association of a stone in the common duct and carcinoma of the head of the pancreas, in which event the gall-bladder might be dilated, or small, thickened, and atrophic or sclerosed.

Sometimes when the gall-bladder contains several stones and its walls are not too tense, distinct gallstone *crepitus* may be elicited by palpation. This is especially valuable diagnostically when the symptoms are not frank.

As already stated, fever is common in cholelithiasis. Of 216 patients, 143 (66.2 per cent.) had fever some time during the course of the disease; 65 (30.1 per cent.) had no fever while under observation; and in 8 (3.7 per cent.) there is no note of the presence or absence of fever. This fever should be correctly interpreted; it is a manifestation of infection, and is not due merely to reflex causes, nervous perturbations, etc., as was at one time thought. It is variable in its manifestations. In many cases, concurrently with the development of the gallstone colic, there is a sudden elevation of the temperature, followed sometimes by an equally sudden fall; the onset of this fever may or may not be associated with chilliness or a definite chill; in other cases the fever is of somewhat longer duration, and is more or less obviously due to an acute cholecystitis or an acute exacerbation of chronic cholecystitis; in other cases the fever is slight and not detected unless diligently searched for, perhaps it is sometimes absent—evidently cases of chronic low-grade infection; in other cases, especially cases of calculus in the common bile duct, the fever is that known as Charcot's intermittent hepatic fever—periodic attacks of chill, fever (103 to 104°), and sweats, accompanied by pain in the epigastrium and the right hypochondrium, recurrence or increase of jaundice, and perhaps nausea and vomiting. This fever is only an aggravation of the types previously mentioned; all are due to biliary infections.

Obstruction of the Cystic Duct.—The calculus is usually lodged in the beginning of the duct or in the termination of the neck of the gall-bladder. The following conditions may ensue: (1) Acute cholecystitis (catarrhal, suppurative, phlegmonous, or gangrenous). (2) Chronic dilatation of the gall-bladder, a condition that follows complete obstruction to the entrance of bile into the gall-bladder, which soon becomes distended with mucus, at first bile-stained, but ultimately quite clear. The gall-bladder content is perhaps partly inflammatory, in the beginning at least, but in many cases the infection gradually subsides, whereas in others it becomes relighted or a new infection and acute cholecystitis ensue. (3) Chronic fibrous or atrophic cholecystitis—the usual sequel of hydrops when the obstruction is not complete and the infection dies out. The gall-bladder gradually becomes reduced in size and may ultimately be reduced to a mass of fibrous tissue, which usually tightly encircles a calculus. When the gall-bladder becomes small and contracted, symptoms may be in abeyance, or they may be those of chronic cholecystitis with or without acute exacerbations, or those of precholecystitic adhesions, etc. Often there is incrustation of the wall with cholesterin and other constituents of gallstones; there may be diverticula which usually contain calculi.

Obstruction of the Common Bile Duct.—There may be one or many obstructing calculi; in the majority of cases there is only one, and this is usually in the lower end of the common duct or the diverticulum of Vater, but it may be in any part of the duct; when there are many calculi they may extend throughout the common duct into the hepatic duct and its branches and into the cystic duct and the gall-bladder. The obstruction may be complete or incomplete, and may or may not be associated with cholangitis. When the obstruction is complete a stone is usually tightly impacted in the common duct; when the obstruction is incomplete the stone may be more or less

fixed, but permit the flow of bile around it, or it may circulate in the common duct and imitate the conditions in a ball valve. A calculus originally tightly impacted in the common duct often, in the course of time, becomes more or less free, in consequence of atrophy or ulceration of the wall of the duct about the stone, or of dilatation of the duct above the stone. Dilatation of the ducts is a common event; it is usually cylindrical, the common duct not infrequently becoming as large or larger than one's finger; the dilatation often extends into the interior of the liver, and may occasion prominence on the surface of the organ; occasionally the dilatation is saccular, the common duct attaining the size of an orange; small saccular dilatations may also occur on the surface of the liver, especially the left lobe. The liver at first is usually somewhat enlarged from accumulation of bile; later atrophy of the hepatic cells ensues, and in consequence of biliary infection, inflammatory phenomena spread from the bile ducts to the adjacent tissue—cholangitis and pericholangitis. This gives rise to some new connective tissue, and the atrophy of the hepatic parenchyma causes a relative increase in the preëxisting connective tissue, so that a condition resembling cirrhosis is induced; but although a form of chronic hepatitis, this is not cirrhosis in an acceptable sense, and it is at best doubtful if biliary stasis can lead to true cirrhosis.

The clinical manifestations of a stone in the common duct vary, depending whether the obstruction is complete or incomplete and whether or not it is associated with infection of the ducts. In the event of complete obstruction without infection, permanent and lasting jaundice is produced, unattended by fever and other signs of local infection. In the event of incomplete obstruction suggestive symptoms are sometimes absent; a stone as large as the terminal phalanx of one's thumb may be present for years without jaundice having resulted. Usually, however, jaundice is present; it lasts a long time, and, varying with the degree of biliary obstruction, is subject to more or less fluctuation, which is very characteristic; and there is fever, periodic, intermittent, remittent, or more or less continuous, indicative of the concomitant biliary infection. This biliary infection is present in practically all cases of incomplete obstruction; it may continue for years without the advent of suppuration, but suppurative cholangitis and cholecystitis may ensue.

Naunyn gives the following as the characteristic signs of a calculus in the common bile duct: "The continuous or occasional presence of bile in the feces; distinct variations in the intensity of the jaundice; normal size or only slight enlargement of the liver; absence of distention of the gall-bladder; enlargement of the spleen; absence of ascites; presence of febrile disturbances, and duration of the jaundice for more than a year." Osler has emphasized the importance of the following signs in the diagnosis of a ball-valve calculus in the diverticulum of Vater (where it occurs most frequently), or in the common bile duct: "(a) Ague-like paroxysms of chill, fever, and sweating—the hepatic intermittent fever of Charcot; (b) jaundice of varying intensity, which persists for months or even years, and deepens after each paroxysm; and (c) at the time of the paroxysm, pain in the region of the liver with gastric disturbance. . . . Pain, which is sometimes severe and colicky, does not always occur. There may be vomiting and nausea. As a rule, there is no progressive deterioration of health. In the intervals between the attacks the temperature is normal."

When the calculus is situated in the diverticulum of Vater it may obstruct the duct of Wirsung, and should the duct of Santorini be unable, on account of non-communication, to divert the pancreatic juice into the duodenum, accumulation of the juice and dilatation of the ducts ensue; furthermore, the common infection present in these cases gradually spreads to the pancreatic ducts, producing pancreatitis, with early enlargement of the organ, especially the head, which may become so hard as to suggest carcinoma; later the organ becomes contracted and fibrosed (interstitial pancreatitis); in some cases pancreatic lithiasis also ensues. Mayo¹ states that in 2200 operations upon the gall-bladder and biliary ducts the pancreas was found coincidentally affected 141 times (6.1 per cent.); since the total number of operations undertaken for disease of the pancreas was only 168, it was found that 81 per cent. of the cases were due to or accompanied by gall-stones. In 268 operations upon the common and the hepatic ducts the pancreas was found diseased in 18.6 per cent., as contrasted with 4.45 per cent. of the cases in which the gall-bladder only was diseased.² In the event of incomplete obstruction of the pancreatic duct, bile may be diverted into it, and lead to the production of acute hemorrhagic pancreatitis, as has been demonstrated by Opie and others.

Complications and Sequels of Cholelithiasis.—Of the many complications and sequels of cholelithiasis, some infectious, some purely mechanical, but most of them infectious and mechanical, the following are the most important: (1) Infectious (inflammatory) lesions of the biliary tract and adjacent viscera; (2) chronic pericholecystic adhesions; (3) biliary fistulæ; and (4) intestinal obstruction.

Infectious(Inflammatory) Lesions of the Biliary Tract and Adjacent Viscera.—It has been pointed out that gallstones are the consequence of infective (low-grade) inflammation of the biliary tract. Having been formed, gallstones participate in a vicious circle and serve to keep alive the infective inflammation to which they owe their origin, in consequence of which and also on account of certain mechanical effects, divers inflammatory lesions of the biliary tract and adjacent viscera ensue. These comprise especially varying grades of acute and chronic, suppurative and non-suppurative, cholangitis and cholecystitis, progressing perhaps to ulceration, gangrene, perforation, etc., to which reference has already been made. The inflammatory phenomena may extend to adjacent structures, and set up local peritonitis (with abscess formation) or a more widespread peritonitis, pylephlebitis, pancreatitis, etc., or, in consequence of the adhesions to which they may give rise, they may seriously compromise the functional activity of the gastro-intestinal tract; or progressing to necrosis and suppuration and ulceration they may lead eventually to the formation of fistulæ.

Chronic Pericholecystic Adhesions.—Adhesions are frequently encountered in the upper abdomen; of 216 patients, 123 (56.6 per cent.) had adhesions about the gall-bladder; 22 (10.1 per cent.) had no adhesions; and of 71 (33.0 per cent.) there is no note of the presence or absence of adhesions (probably none were present). These adhesions represent the sequels of past subacute and chronic, sometimes acute, inflammation of the organs of

¹ *Journal of the American Medical Association*, 1908, I, 1161.

² Consult also Mayo Robson, *Surgery, Gynecology, and Obstetrics*, 1908, vi, 29; and Maugeret, *Cholécysto-pancréatite*, Paris, 1908.

the upper abdomen, of which the biliary tract is the chief; whence they are a direct result of biliary infection.

The conditions are quite analogous to those in and about the vermiform appendix. Should the biliary infection be acute and virulent, the bacteria or their toxins penetrate the wall of the gall-bladder or the biliary ducts and engender a fibrinous exudation, a more or less local peritonitis. In the event of the later development of suggestive clinical symptoms, much diagnostic significance attaches to a history of such past infections. In the subacute and chronic infections, however, the formation of adhesions goes on less obtrusively, often entirely latently, whence the symptoms that develop later are commonly misinterpreted. In consequence of milder infections the less virulent toxins give rise to less inflammatory phenomena—sufficient, however, often to lead to more or less widespread adhesions. All gradations are encountered; frequently these are quite localized and very delicate (gall-spiders, resembling spider webs, they have been aptly called by Morris); in other cases they are quite widespread and very dense.

The clinical recognition of these adhesions is sometimes a matter of difficulty, but it is perhaps not impossible in the majority of cases. It is essential, in the first place, that we recognize that many of the cases of so-called “stomachache,” “biliousness,” indigestion, etc., in reality have an anatomical basis. Many of these cases are due to an unsuspected or latent gastric or duodenal ulcer, definite cholecystitis, chronic pancreatitis, etc.; in other cases they are due to adhesions. We must study attentively patients who complain of general ill-health, with more or less ill-defined gastric or epigastric symptoms. Attacks of such symptoms may come on periodically without definite cause; they may last a few days, and cease quite as causelessly. Now and then the attack may be ushered in with chilliness (rarely a definite chill), and may be attended by a little fever. Examination may reveal some tenderness in the epigastrium, perhaps a little to the right of the median line, toward the region of the pylorus or the gall-bladder, and some rigidity of the overlying muscle. Deep pressure is sometimes very painful. Later, should the pylorus or the duodenum become obstructed, the commoner manifestations of dilatation of the stomach supervene. Especial diagnostic importance attaches to intractability of the symptoms and a history of past infection of the biliary tract. In the event of suspecting such adhesions, we must exclude other factors that may cause similar symptoms, remembering, however, that in many cases symptoms supposedly due to disease of the stomach or intestine are due to disease of the gall-bladder or adhesions of the upper abdomen.

Biliary Fistulæ.—Fistulous communications between the biliary tracts and adjacent viscera as well as the exterior of the body frequently result from cholelithiasis and the associated inflammatory and ulcerative processes. Courvoisier in a study of 499 cases found that fistulous communications had been established as follows: between different parts of the biliary passages in 8; into the portal vein in 5; into the peritoneal cavity in 70; into peritoneal adhesions in 49; into the retroperitoneum in 3; with the stomach in 13; with the duodenum in 83; with the jejunum in 1; with the ileum in 1; with the colon in 39; with the urinary tract in 7; with the pleura and lung in 24; and externally in 196. The external fistulæ appear to be disproportionately numerous since by their very nature they command attention, whereas the internal fistulæ frequently are not detected. In external fistulæ

the opening may be near the normal situation of the gall-bladder, near the umbilicus (being directed thither by the falciform ligament), in the right iliac fossa, in the thigh, etc. Biliary gastric and intestinal fistulæ are often unsuspected; sometimes they are first revealed by the discharge of gallstones with the fæces (without concomitant gallstone colic), or by the sudden disappearance of long-standing obstructive jaundice, or by the onset of intestinal obstruction (gallstone ileus). Bronchobiliary fistulæ lead to cough, dyspnœa, and the expectoration of pus, bile, and perhaps gallstones. Schlesinger¹ and Eichler² recently have added to the cases and there are now 47 cases on record, of which 28 were due to gallstones.

Intestinal Obstruction.—Intestinal obstruction is a rather rare event that may follow ulceration of a gallstone from the gall-bladder into the duodenum or, less commonly, the colon. It is doubtful if a gallstone that has been able to pass through the biliary ducts and the ampulla of Vater into the duodenum is large enough to obstruct the intestine; in some cases, however, a comparatively small stone may become arrested at the ileocecal valve; it may set up inflammation and localized spasm of the intestine with consequent obstruction; it may become increased in size after it reaches the intestine (enterolith); or it may become arrested in a part of the intestine narrowed by inflammatory or neoplastic disease, etc. The ratio of gallstone ileus to other forms of intestinal obstruction varies between 1 to 13 (Fitz) and 1 to 45 (Barnard). Recently, Lesk³ has collected 148 cases, of which 109 (73.5 per cent.) occurred in women and 39 (26.5 per cent.) in men. Only 6 of the patients were less than forty years of age; most of them were beyond fifty. In 104 of Lesk's cases the obstruction was in the ileum in 62 (59.6 per cent.), in the jejunum in 17 (16.3 per cent.), in the small intestine (without definite statement) in 13 (12.5 per cent.), in the colon in 6 (5.7 per cent.), in the duodenum in 5 (4.8 per cent.), and in the rectum in 1 (1 per cent.). In 26 of the 62 cases in which the obstruction was in the ileum it was near the ileocæcal valve. The diagnosis rests upon the advanced age of the patient and a past history of gallstones or other manifestations of biliary infection; in some cases the gallstone causing the obstruction and the surrounding or adjacent tissues form a palpable mass in which the gallstone may be distinguishable. In most cases, however, the cause of the intestinal obstruction cannot be diagnosed with certainty until it is seen. Sometimes the obstruction is relieved spontaneously, but at least one-half of the patients die unless the obstruction is relieved by surgical procedures. The operative mortality also is very high (about 45 per cent.), which is due largely to delay, but also to the advanced age of the subjects.

Diagnosis.—The diagnosis of cholelithiasis comprises the recognition of the gallstone colic and of the many and varied associated lesions and sequels. Severe pain in the epigastrium or the right hypochondrium, radiating around the chest or to the right scapula, accompanied by nausea and vomiting and enlargement and tenderness of the gall-bladder, and followed by jaundice and the detection of a gallstone in the fæces, are characteristic of the passage of a gallstone; but jaundice is by no means always present and the other symptoms may be provoked by cholecystitis in the absence of a gall-

¹ *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, 1906, xvi, 240.

² *Ibid.*, 1906, xvi, 550.

³ *Deutsche Zeitschrift für Chirurgie*, 1908, xcix, 47.

stone; indeed, with increasing experience it becomes often more and more difficult to say whether or not gallstones are present in certain individual cases of obvious chronic biliary infections.

Recourse to the x -rays is seldom of much diagnostic utility, since the cholesterin stones produce scarcely any shadow, usually not more than the adjacent liver; in the rare cases of calcium stones more shadow is cast, but this also is obscured by the shadow of the liver. In a few cases, however, the x -rays afford corroborative if not absolutely trustworthy evidence.

A diagnosis after the event is readily made in many cases, but during the prevalence of the severe pain, one may be unable to determine the cause of the patient's suffering. A few whiffs of chloroform or a hypodermic injection of morphine may so relieve the patient as to permit of an examination, in which event tenderness and perhaps enlargement of the gall-bladder may be elicited. The severe pain of gallstone colic may be imitated by renal colic or intestinal colic; but in renal colic the pain is usually in the back, loin, or groin, and radiates down the ureter to the testicle, which may become retracted; strangury or frequent micturition is common; the kidney is usually tender (the tenderness being in the loin rather than anteriorly in the region of the gall-bladder); and the urine subsequently contains blood and a proportionate amount of albumin, perhaps crystals or a calculus, and pus and epithelium from the pelvis of the kidney; and it does not contain bile. Intestinal colic is usually due to some more or less obvious cause; the pain is usually about the umbilicus, it radiates in different directions, remits and intermits, and subsides with the removal of the exciting cause, often with belching or the discharge of fæces or gas or mucus by the bowel; and there is no localized tenderness in the region of the gall-bladder. In lead colic there is, in addition, the history of exposure to the poison, a blue line on the gums, anæmia, basophilic degeneration of the erythrocytes, and perhaps other present or past evidences of the intoxication, such as persistent abdominal discomfort or pain and constipation, wrist-drop, etc.

When the symptoms, especially the pain, are less acute, and do not merit the designation gallstone colic, difficulty may be experienced in differentiating disorders of the stomach. Much assistance will be obtained by bearing in mind that the case of so-called chronic indigestion that is not benefited by several months of well-directed therapy commonly has an anatomical basis. Gastric or duodenal ulcer is suggested by dependence of pain upon taking food or its occurrence as the chyme passes into the duodenum (which sometimes obtains also in cholelithiasis), by tenderness in the median line or slightly to the right, and posteriorly on the left side near the vertebræ, hyperchlorhydria, hæmatemesis, etc., and the absence of jaundice and intermittent hepatic fever; prominence of nausea and vomiting is more significant of cholelithiasis. Pericholecystitis and perigastric adhesions are often the cause of so-called chronic indigestion, and may give rise to pyloric obstruction and gastric dilatation.

Malignant disease of the gall-bladder usually follows cholelithiasis, but so insidious, as a rule, is its onset that it is commonly not suspected until the advent of loss of flesh and strength and the progressive development of cachexia. In some cases of more or less apparent cholelithiasis the diagnosis of carcinoma is favored by the detection of progressive nodular enlargement of the gall-bladder, and the progress of the disease is shown in some cases by the occurrence of metastasis to the liver, ascites, etc. In the event

of jaundice, its persistence unchanged suggests malignant disease of the ducts or pancreas, etc., although it may be due to a stone tightly impacted in the common duct; but there is less impairment of the general health than in carcinoma; in the majority of cases of gallstones in the ducts, as contrasted with malignant disease or catarrhal cholangitis, there is periodic variation in the intensity of the jaundice (of much diagnostic significance) and fever (intermittent hepatic fever).

Prognosis.—The prognosis, as a rule, depends more upon the associated conditions than upon the mere presence of the gallstones. The prognosis in gallstone colic is virtually that of a more or less acute cholecystitis; the outlook as regards permanent health depends upon the chronic gall-bladder infection. In almost all cases of gallstone colic the acute symptoms subside spontaneously after several hours or several days; rarely a patient may die, it is said from shock and cardiac failure; occasionally rupture of the gall-bladder ensues and gives rise to infectious peritonitis and a fatal issue; and in other cases a stone impacted in the neck of the gall-bladder or the cystic duct and the attendant infection and interference with the blood supply may, after the lapse of several days, lead to severe suppurative, phlegmonous, or gangrenous cholecystitis.

A first gallstone colic is sometimes the last and the infection subsides. Usually, however, there are subsequent attacks, if not of severe colic, of more or less disturbance with digestion, and general impairment of health, due to the toxic absorption, etc.; some subjects pass into chronic invalidism from unrecognized chronic biliary infections. The likelihood of continuation of symptoms is increased by the duration of symptoms in the past; that is, those already ill for some time are likely to continue ill, and the lesions to progress. The detection of faceted stones in the fæces suggests that other stones are still undischarged; the detection of rounded or ovoid stones in the fæces, together with subsidence of the local symptoms, arouses the hope that there was but a single stone. The occurrence of intermittent hepatic fever, especially of severe ague-like paroxysms, renders the prognosis rather bad, since it shows active infection, which, although it may persist for years without suppuration, may nevertheless lead to serious consequences. The prognosis as to final recovery of health is bad in pericholecystic adhesions, fistulæ, and intestinal obstruction without operative intervention.

Treatment.—The intelligent treatment of cholelithiasis presupposes a clear conception of (1) the pathology of the disease, of its pathogenesis, pathological anatomy, and natural tendencies, and (2) of the means of treatment at our command and the objects attainable by their use. To attempt by internal medication to dissolve a gallstone that is insoluble; to cause the passage through the biliary ducts of a gallstone when the ducts are impassable to a stone of its size; to attempt to cure supposed gastric symptoms by measures directed to the stomach when the cause of the symptoms is adhesions about the gall-bladder, and to cause the solution of these adhesions when, perchance, they have been diagnosed, is as futile as it is irrational.

Prophylaxis.—A knowledge of the etiology of cholelithiasis suggests certain measures of prophylaxis. Although we may be quite unable to prevent the causative infection in the individual, we may advise useful measures. The fact that gallstones are most common in women, in the obese, in those that lead a sedentary life, eat too much, are constipated, and addicted to

alcohol, suggests the correction of certain very obvious etiological factors. Women who have been repeatedly pregnant and have lax abdominal walls should wear proper supports; but women, whether they have been pregnant or not, should avoid tight lacing. Men and women who lead a sedentary life should take properly regulated exercise; they should practise deep breathing (to promote the movements of the diaphragm), bathe frequently, be much in the open air, etc.; that is, they should apply to themselves the general principles of hygienic living. Constipation should be overcome, and the diet should be such as will be presently pointed out. If these measures do not prevent the formation of gallstones in those who have or have had a biliary infection, they will at least promote a general well-being, tend to reduce the concomitant biliary infection, and perhaps obviate some of the serious consequences of cholelithiasis.

When gallstones have formed, the main indications for treatment may be said to be: (1) To cause solution of the stone or stones; (2) to cause the discharge of the stone or stones; and (3) to treat the complicating infection.

Solution of the Gallstones.—As regards the solubility of the gallstones Naunyn¹ states that we have to consider cholesterin, bilirubin-calcium, calcium carbonate, and calcium phosphate. Of these, bilirubin-calcium and the inorganic salts of calcium are insoluble in the bile; cholesterin, however, is quite soluble in the bile, and even in conditions of disease the bile scarcely if ever contains so much cholesterin that it cannot take more in solution. We have, therefore, to admit the possibility of cholesterin stones being dissolved. In fact, there is some experimental evidence that goes to prove that not only *in vitro*, but also *in vivo*, cholesterin stones may be dissolved. Naunyn quotes Labes' experiments of thirty years ago, in which he introduced gallstones into the gall-bladder of dogs, and, killing the animals two months later, found that partial solution of the stones had occurred. Bain² also has published the results of some studies that show the same possibility. In these experiments, however, the calculi were introduced into normal gall-bladders (or, as in one or two of Bain's experiments, into gall-bladders in which a cholecystitis has been artificially produced). The conditions are quite different in the human subject affected with gallstones—in which one has to deal not only with the stones, as such, but also with the subacute or chronic lithogenic catarrh—the direct cause of the gallstone formation. It is thus exceedingly doubtful whether spontaneous solution of gallstones ever occurs in the human subject—indeed, whether it is at all possible. Naunyn states that in studying one thousand cases of cholelithiasis he has seen trustworthy evidence of the solution of the stone in perhaps ten cases (1 per cent.). Although he does not deny that the solvent action of the bile may be a contributing factor, he inclines much more to the belief that disintegration is due to the action of bacteria, and for the reasons: that he has found this disintegration, not in cases of a single stone, but in cases of numerous stones; that the process involved many stones; that the solution was not circumferential, as it would be in the event of solution in a dissolving menstruum, but was circumscribed at the periphery and extended deep into the calculi; and that the disintegration went hand-in-hand with the deposition in the stones of inorganic

¹ *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, 1905, xiv, 537.

² *British Medical Journal*, 1905, ii, 269.

calcium (phosphates and carbonates), certainly due to bacterial activity. The reasons for the non-occurrence of solution of the stones are: (1) and most important, the fact that on account of the presence of the stones the activity of the walls of the gall-bladder is compromised, it cannot empty itself completely, and the desirable irrigation of the gall-bladder with fresh bile does not occur; and (2) the fact that on account of the associated catarrh of the biliary tract the bile contains more cholesterin than is normal and thus takes up an additional increased amount.

Naunyn furthermore points out that even should it be possible to cause disintegration of the stones in the gall-bladder, it is exceedingly doubtful how much good would be thereby accomplished—since in this event the residual particles of the disintegrated calculi would remain in the gall-bladder and form the nucleus of new calculi. These are formed by the cementing together or incrustation of the disintegrated particles by newly precipitated bilirubin-calcium, the formation of which is favored by the persisting infectious inflammation of the mucous membrane, which gives rise to a secretion rich in calcium.

This reasoning is well borne out by clinical experience, since it is exceedingly doubtful whether, with any means at our command, we can cause solution of gallstones in the gall-bladder. We now know that the thousands of gallstones said to have been passed by the bowel after the administration of olive oil are merely masses resembling gallstones in outward appearances (saponified oleic acid). There is also no reason to believe that olive oil, derivatives of olive oil and other oils, turpentine, ether, etc., have any influence whatever in dissolving gallstones in the biliary tract.

Discharge of the Gallstones.—Gallstones may be discharged from the biliary tract by way of the ducts, or by way of fistulæ. Many stones rarely pass through the ducts, although single stones sometimes do; large stones, if discharged, practically always leave the gall-bladder by devious tracts and fistulæ. In some cases the gallstones have been entirely latent until they appear in the stools, in which event, if the stone is large, we must assume the formation of a gall-bladder-colonic fistula, since large stones, having ulcerated into the small intestine, practically always cause noteworthy symptoms in their downward passage. In the event of a single stone only being present, the possibility of cure is thus presented, but the possibility is too remote for serious consideration. It is an occurrence which should not be hoped for, not awaited or sought by medical means, even were such at our command; it cannot be considered an object of rational treatment.

Treatment of the Biliary Infection.—The one worthy object is to restore the latency from which the gallstones have been awakened, in other words, to control the infection at the basis of gallstone activity. This must also be the object of medicinal or internal treatment of cholecystitis in the absence of gallstones. The infection of the biliary tract is what the physician has to treat. The surgeon may remove the stones and also treat the biliary infection. The physician, however, in many cases, may lead his patient into a condition of virtual cure, that is, latency or sterility of the biliary tract; but he must recognize his limitations, and by a judicious balancing of his abilities and limitations not subject his patient too long to a useless medicinal treatment when early surgical intervention may restore him to health, whereas delayed it may not only add to the miseries of a miserable existence, but actually hasten the final termination.

The manifestations of the biliary infection calling for treatment may be acute, that is, gallstone colic, or chronic.

The *pain of gallstone colic* is usually so severe as to require the administration of morphine, which not only relieves the pain (and mitigates the attendant shock), but, by relaxing the spasm of the biliary musculature, favors the exit of bile from the tense gall-bladder and the transit of the calculus through the ducts (if it be already therein). When required, morphine should not be withheld, but more than is necessary should not be given. Usually, $\frac{1}{4}$ grain with $\frac{1}{50}$ grain of atropine or $\frac{1}{100}$ grain of nitroglycerin suffices; sometimes after the lapse of fifteen to thirty minutes a second smaller dose may seem necessary, but in these circumstances a few whiffs of chloroform, or spirit of chloroform or of ether (1 dram), to which tincture of belladonna (5 to 10 minims) may be added, will usually carry the patient through the painful period until the morphine has had time to act; or, and this is often attended with excellent results, an additional dose of atropine ($\frac{1}{100}$ grain) may be administered hypodermically. Should resort be had to chloroform inhalation, one should not induce complete anæsthesia in the hope of thus relaxing the biliary musculature and freeing the calculus: such objects are not thus achieved.

The use of coal-tar products for the relief of the pain is ill advised, since not only are they comparatively inefficacious, but they also add to the depression of an already much depressed patient; the salicylates and olive oil (even when tolerated) also are of no value during the acute stage, although olive oil by inducing vomiting seems sometimes to assist in dislodging the stone. Sometimes also it may relieve an associated hyperchlorhydria.

In many cases relief may also be afforded by hot fomentations over the region of the gall-bladder and by lavage of the stomach with water as hot as can well be borne. All food should be withheld during the continuance of the acute symptoms; indeed, the nausea and vomiting lead the patient usually to decline food. Vomiting ceases with the cessation of the pain, so that the best treatment for the vomiting is that that tends to relieve the pain and the lesions upon which it is founded. Should the vomiting persist and be manifest especially by ineffectual retching, resort should be had to lavage: if it has not been previously used, sodium bicarbonate may be added to the hot water. The hot full bath may also be resorted to at the very beginning of the attack, with hope of excellent result. In other cases the vomiting may be relieved by bismuth, carbolic acid, creosote, diluted hydrocyanic acid, cerium oxalate, sips of hot water and brandy, champagne, etc. In some cases the pain leads to extraordinary depression of the patient, which if it is not relieved by the morphine, should be met with whisky or brandy in hot water, or the hypodermic use of camphor.

Upon the subsidence of the pain, as well as in the absence of a definite gallstone colic, the medical treatment of cholelithiasis is that of the chronic biliary infection; this is of the greatest importance in many cases, and it finds also a special field for service after surgical intervention. By promoting the free flow of bile one may effect the discharge of infecting bacteria and their toxins from the biliary passages and prevent their ascent to the smaller ducts, and by the constant irrigation thus produced, as well as by other measures, one may allay the inflammatory phenomena. Ultimately, the biliary tract may be rendered sterile, and should the inflammatory phenomena subside completely a condition of latency, quiescence of the

gallstones, results. In rare cases this latency is permanent, and the patient experiences no further complaints. In most cases a low-grade chronic catarrh of the gall-bladder persists; in other cases the gall-bladder readily again becomes infected, and the patient in consequence is continuously or intermittently ill. Should gallstones be passed by the bowel, one must remember that all the gallstones harbored by the patient are seldom, if ever, thus passed, and that even were this the case, the persisting catarrh of the gall-bladder is quite certain to give rise to the formation of new stones.

The objects of medical treatment are reduction of infection and relief of congestion and inflammation; these are sought by the pursuit of measures that tend to lessen congestion and the avoidance of measures that tend to promote it, by flushing the biliary tract, and by establishing biliary asepsis. The prophylactic measures already mentioned are of the greatest service, especially exercise, regulation of the diet, and overcoming constipation. That exercise which tends to general muscular activity and promotes movements of the diaphragm is preferable, but it should be prescribed only after a careful study of the general bodily conditions, especially the cardiovascular system. After parturition efforts should be made to restore the abdominal parietes to their normal tone; massage may render excellent service.

The *diet* should be carefully regulated. In character and amount it should be such as may be readily digested, and not overtax the resources of the liver and promote congestion; and the time intervals between meals should be such as tend to frequent and free emptying of the gall-bladder. Cholelithic subjects should have a sufficient but not an excessive diet; in character it should be based less upon the mere presence of gallstones than upon the attendant gastro-intestinal catarrh, constipation, and jaundice (if present). In general a mixed diet is the best; this should contain considerable and varied proteid, which is believed to promote an abundant supply of bile acids and to stimulate the flow of the bile. In the presence of jaundice, proteids and readily digested carbohydrates should be advised, and the fats should be much reduced or excluded for a time, since they are ill digested in the absence of bile; but in the absence of jaundice it is unnecessary to prohibit fat on the mistaken apprehension that a fatty diet tends to increase the cholesterin in the bile. Foods which set up or continue a gastro-intestinal catarrh in the individual should be avoided. Naunyn has said that a full meal is an excellent cholagogue, so that with the hope of preventing stagnation of the bile, an additional meal may be given at bedtime, unless in some way contra-indicated. In most cases it is wise to prohibit the use of alcohol.

The utility of large amounts of water, especially of alkaline mineral waters containing sodium salts, is unquestionable. Since they have no solvent action on the gallstones, and do not influence the alkalinity of the bile, their beneficial effect is probably on the associated biliary and gastro-intestinal catarrh. There is some experimental evidence that these waters do not directly stimulate the flow of bile, and that the excessive amount of fluid does not dilute the bile, but this doubtless results indirectly, the removal of biliary constituents from the intestine leading to renewed formation; and an insufficient supply of water tends to inspissation and stagnation of the bile.

The treatment can often be best carried out at one of the well-known spas, but equally good results may be obtained at the patient's home,

although they are unlikely. Of Carlsbad or similar water, from a half to a pint should be taken hot on an empty stomach, one half to one hour before breakfast, and a similar amount in the middle of the afternoon; in some cases, the best results follow smaller amounts a half-hour before each meal and on retiring at night. It is wise to begin with small doses and gradually increase; the amount and the frequency of administration should be gauged by the result, particular attention being paid to free evacuation of the bowels. The saline cathartics, magnesium or sodium phosphate, may be used instead of the natural waters. The effect on the bowels may be enhanced by large injections of hot water (in some cases cold water).

Many drugs have been advocated in the treatment of cholelithiasis from time to time, but few have withstood the test of experience. Cholagogue properties have been ascribed to many that need not be mentioned; salicylic acid and its preparations and preparations of the bile itself are efficacious. Salicylic acid seems to augment the flow of bile by stimulating its production, and since it also possesses antiseptic properties and is excreted with the bile, it exerts a most desirable influence on the biliary infection. No preparation is better than chemically pure salicylic acid; it may be combined with sodium bicarbonate or sodium benzoate; or the salicylates or other preparations of salicylic acid may be given. Crowe¹ recently has demonstrated that hexamethylenamine is excreted in the bile and the pancreatic juice, directly through the wall of the gall-bladder, etc., and that, especially after single large doses (75 grains within twenty-four hours), it appears in the bile in such amount as to exert a decided bactericidal action. Osgall also stimulates the secretion and outpouring of bile, and thus favors drainage of the biliary tract; it should be administered as salts of the bile acids, such as sodium glycocholate (10 to 15 grains daily). Ammonium chloride also is sometimes extolled for its effect upon inflamed mucous membranes; by promoting the free flow of thin bile it may assist in the much desired flushing of the biliary tract.

Surgical Treatment.—In many cases medication is altogether inefficacious, and resort must be had to surgical intervention. The important question to decide is, When should an operation be undertaken? The writer is by no means prepared to say that the diagnosis brings with it the indication for surgical intervention. The multiplicity of the pathological lesions and of the clinical manifestations, and their course uninfluenced by operation, the general health of the gallstone subject, the hazard of an operation, and the personal inclinations of the patient often make us pause.

In many cases the question of early operation scarcely admits of discussion. These are cases in which there is acute cholecystitis (with or without cholangitis—jaundice), with evidences of severe infection, and in which the symptoms and local signs, instead of abating, become more pronounced: these comprise cases of acute cholecystitis developing during or after an attack of typhoid fever, as well as during the course of previously latent or manifest cholelithiasis, cases in which the diagnosis of severe suppurative or gangrenous cholecystitis with perforation of the gall-bladder or severe local or generalized peritonitis, seems warranted, and in which impending if not actual infection of the hepatic ducts is not unlikely. The

¹ *Johns Hopkins Hospital Bulletin*, 1908, xix, 109.

risk attending the progress of the disease in such cases is more than the hazard of the operation; indeed, without operation many would unquestionably soon terminate fatally. Happily, however, most of the acute infections of the biliary tract subside spontaneously or under treatment. In general, it is much wiser that the acute manifestations should subside without operation—which, should it be deemed desirable later, may then be undertaken with much more reasonable hope of ultimate success.

Immediate operation is imperative also in the event of acute intestinal obstruction in a cholelithitic subject, or in a person in whom adhesions have been suspected. Operation, although not necessarily always immediate, is indicated in all cases of persistently enlarged gall-bladder, whether the enlargement is due to simple hydrops, empyema, or gallstones.

The question of when to operate in cases of gallstones that have become or have been active must be decided in each individual case; but one should bear in mind that, as a rule, it is not the many stones in the gall-bladder that do the most damage, but the one or two stones that get into the ducts, that the special risk attending operations for gallstones increases with delay, and that whereas gallstones are readily removed from the gall-bladder, their removal from the ducts is commonly attended with serious difficulties and is sometimes impossible. The time of election for operation, therefore, is before the stones have entered the ducts. This is by no means always possible, or, indeed, desirable. Thus one should not advise the removal of stones that are and always have been quiescent. If, however, the gall-bladder is enlarged and the stones have given warnings—local discomfort and distress—operation should be undertaken. Nor would the writer insist upon operation after a single short successful gallstone colic (so-called), but he would by no means be averse to it. Two, certainly three, attacks should be looked upon as a positive indication for operative intervention. In the event of hesitation on the part of the patient, the pathological changes and the likely course of the disease should be explained, and he should be required to make the decision for or against operation and assume the responsibility.

In impaction of a gallstone with chronic jaundice, operation is called for; personal conviction is for early rather than delayed intervention. The risk of gallstone operations is the risk of delay, since with delay serious infections of the biliary tract supervene, the tendency to pronounced hemorrhage increases with the persistence of the jaundice, the general nutrition of the patient fails, he becomes less able to withstand the vicissitudes of the operation, and stagnation of the bile leads to dilatation of the ducts and seriously compromises the functional activity of the liver cells. The decision as to the time of operation must be made for each individual patient, but there scarcely seems any advantage attending a delay beyond one or two weeks, and this, too, in full knowledge of the fact that some patients have recovered without operation after a much longer period. Finally, operation is indicated in those obscure cases in which with reasonable certainty the presence of pericholecystic, perigastric, or periduodenal adhesions may be surmised.

In each of the foregoing classes of cases operative measures attain that which is impossible of attainment, or attainable only with difficulty by medicinal measures—whence surgical measures find their justification. They find additional justification in the fact that early intervention tends to prevent the development of certain complications and sequels. An

operation, however, should not be lightly undertaken in persons suffering with disorders of nutrition, or in those with faulty kidneys, etc; the risk attending the operation is too great and the recuperative power of the patient too much reduced to expect from operation a great deal more than can probably be attained by well-directed medicinal, hygienic, and dietetic treatment, aside from the fact that in the one instance the patient will remain alive although indisposed, while in the other he may be much longer dead. Finally, the fact that the operative results are not always what were hoped for is not in itself a contra-indication to operation; on the contrary, these untoward results are often attributable rather to the fact that in many long-delayed cases the anatomical lesions are such as to be almost if not quite irremediable by any and all means at our command.

CARCINOMA OF THE GALL-BLADDER AND BILIARY DUCTS.

Carcinoma of the gall-bladder and biliary ducts may be primary or secondary. The secondary cases are of comparative rarity and of little clinical importance; usually the lesions constitute a subordinate part of a general carcinomatosis. The metastases in the gall-bladder consist of nodules of varying size, rarely of a more diffuse infiltration on or just beneath the peritoneum; the mucosa is seldom involved. The biliary ducts are much less rarely implicated, although occasionally invaded from without in carcinoma of the pancreas, stomach, etc. These secondary growths form usually only an insignificant part of the general metastasis, and can scarcely be recognized clinically, unless the irregular, nodular enlargement of the gall-bladder becomes conspicuous, or the cystic or common bile duct becomes obstructed; in the first of these two events hydrops of the gall-bladder may ensue, and in the other, chronic jaundice, which only with difficulty can be distinguished from other types of chronic jaundice.

Primary carcinoma of the gall-bladder and the biliary ducts is a tolerably frequent disorder—three to four times as common in the gall-bladder as in the biliary ducts. At the necropsy it is often difficult to determine definitely where the lesion began, but it seems desirable to exclude from the cases of primary carcinoma of the biliary ducts those in which the new-growth developed from the fine intrahepatic ducts; these are virtually cases of carcinoma of the liver. When the extrahepatic ducts are involved, the disease may spread along the ducts and involve the gall-bladder, the pancreas, the omentum, etc., or primary disease in any of these structures may spread to the ducts. Thus carcinoma of the gall-bladder and of the cystic duct is virtually one disease, although sometimes it may be differentiated; carcinoma of the gall-bladder and of the pancreas may spread to the biliary ducts, and vice versa; carcinoma of the gastrohepatic omentum may originate in the biliary ducts; and in some cases so widespread is the new-growth and so dense and cicatricial the adhesions that carcinoma, if at all suspected, can only with the greatest difficulty be referred to its true point of origin. In some cases the condition is thought to be inflammatory, and only a microscopic examination will disclose its real nature. With care, however, most cases can be correctly interpreted. Rare cases of sarcoma of the gall-bladder and biliary ducts have been described, but they are only of pathological interest; clinically they cannot be distinguished from carcinoma.

Etiology.—*Primary carcinoma of the gall-bladder* is a fairly common disease. In 1889 Musser¹ collected 100 cases; in 1901 Fütterer² collected 268 cases. The disease is much more common in women than in men; in Musser's cases there were 75 females to 23 males (3 to 1); in Fütterer's, 204 females to 52 males (4 to 1). It is especially prevalent in advanced life; the average age in Fütterer's cases was fifty-eight years. An important etiological question is the interpretation of the relationship between carcinoma of the gall-bladder and cholelithiasis. Frequently they are associated: in 69 per cent. of the cases (Musser), in 78 per cent. (Fütterer), in 81 per cent. (Winton), in 85 per cent. (Zenker), in 91 per cent. (Courvoisier), in 95 per cent. (Siegert), and in 100 per cent. (Janowski). Rolleston, from a study of many statistics, estimates that carcinoma of the gall-bladder occurs in from 4 to 14 per cent. of cases of cholelithiasis. At 262 operations on the gall-bladder and biliary ducts done by Dr. Deaver at the German Hospital, Philadelphia, carcinoma was found in 6, of which 5 had also gallstones.

Carcinoma of the extrahepatic biliary ducts has been attentively studied by Rolleston, who has collected 80 cases. Of 75 of these, 44 (58.7 per cent.) occurred in males and 31 (41.3 per cent.) in females. In 52 (71 per cent.) of 73 cases the patient was beyond fifty years of age; extremes of age were eighty-one (a woman) and twenty-nine years (a man). In contrast with carcinoma of the gall-bladder, gallstones are much less frequently associated with carcinoma of the biliary ducts; in 62 of Rolleston's cases, gallstones were absent in 39 (63 per cent.) and present in 23 (37 per cent.).

Carcinoma of the ampulla of Vater (choledochopancreatic duct) also has been studied by Rolleston, who has collected 16 cases; of these, 13 were in males and 3 in females; the average age was 55.6 years in both sexes, the extremes being thirty-four and eighty-one years. Since gallstones were present in only 2 of the 16 cases, it is likely, as Rolleston maintains, that there is no relationship between gallstones and carcinoma of the ampulla of Vater.

Pathology.—*Carcinoma of the gall-bladder* most commonly involves the fundus, which is said to be due to the fact that, being the most dependent part, the fundus is especially subject to irritation by a gallstone; second in point of frequency of growth is the neck of the gall-bladder or the beginning of the cystic duct—attributed to the attrition of impacted gallstones; third, the growth may be situated anywhere in the gall-bladder or involve the entire organ. In 45 of Fütterer's cases the growth was situated at the fundus in 17, at the neck in 13, on the posterior wall in 8, and on the anterior wall in 7. Two types of growth may be distinguished: that which projects into the lumen, a villous or cauliflower-like growth, and is believed by some to originate as a papilloma, and that which infiltrates the wall of the gall-bladder, and is believed by some to originate as an adenoma. In reality the two varieties cannot be differentiated; the one growth may exhibit appearances significant of both types of growth. In the early stages the growth is more or less circumscribed, but as the disorder advances, infiltration usually becomes more marked and gives rise to irregular nodular enlargement of the gall-bladder. In other cases, especially when the growth begins at or near the cystic-duct extremity, the outlet of the gall-bladder becomes obstructed, and the gall-bladder becomes distended with fluid, forming a

¹ *Boston Medical and Surgical Journal*, 1889, exxi, 525, 553, 581.

² *Ueber die Aetiologie des Carcinoms*, 1901.

tense, pear-shaped tumor. With the progress of the disease, more or less extensive infiltration of the gall-bladder usually ensues; in some cases it may be represented by a mass of variably dense and softened tissue, in which the identity of the gall-bladder may have become completely lost; in some cases its lumen can be determined only by the presence of one or more gallstones; in some cases colloid degeneration is encountered. Extension to the liver ensues in more than 50 per cent. of the cases, from direct growth into the adjacent liver, or by extension along the biliary ducts, by way of the lymphatics in the wall, or through the lumen of the ducts. Adhesions between the carcinomatous gall-bladder and adjacent organs are common. When the pylorus or the upper part of the duodenum is involved, the condition is virtually that of pyloric obstruction, and may simulate carcinoma of the stomach. When the colon is involved, intestinal obstruction may be caused. In rare cases the gall-bladder perforates into the colon; less rarely a fistulous communication may be established with the duodenum, the stomach, or the exterior by way of the abdominal wall.

Microscopically, carcinoma of the gall-bladder may present the appearances of cylindrical- or cuboidal-cell carcinoma, and it has been suggested that one type originates from the surface epithelium, the other from the deeper cells lining the mucous glands; the distinction cannot always be maintained, since transitions from one to the other type occur in the same growth. The designation glandular carcinoma sufficiently well describes almost all the tumors. Retrograde changes sometimes occur. A few cases of the so-called squamous carcinoma have been described.

In the 80 cases of *carcinoma of the biliary ducts* collected by Rolleston the situation of the growth was as follows: In the common bile duct, 33 (lower end 21, midpart 11); at the junction of the common bile duct, cystic duct, and common hepatic duct, 25; in the common hepatic duct, 18; in the right or left hepatic ducts, 3 (the last two classes constitute the so-called juxta-hepatic cases); in the cystic duct, 1; and in the cystic duct and the lower end of the bile duct, 1. Usually the growth infiltrates the wall of the duct and forms a firm annular stricture; occasionally it extends in the wall of the ducts for a considerable extent, and transforms them into thick rigid tubes; in some instances the growth projects considerably into the lumen, and produces obstruction rather than annular stricture. Complete biliary obstruction is produced during life, due, in part, probably, to added muscular spasm, since after death the stricture does not always appear to be absolutely impervious.

Above the growth the biliary ducts are dilated, sometimes to such an extreme degree as to allow a finger or a thumb to be introduced. When the growth is in the common bile duct the gall-bladder is distended, except it be bound down and retracted on itself in consequence of former cholelithiasis; when the growth is in the common hepatic duct, the gall-bladder is nearly always small, except in the rare instances in which it may be distended with mucus from concomitant obstruction of the cystic duct, or may be occupied by a number of gallstones; when the growth is situated at the junction of the cystic, hepatic, and common bile ducts, the gall-bladder, as a rule, is not enlarged, but from irregularities and variations in the degree of obstruction to the different ducts, corresponding differences in the condition of the gall-bladder are met with.

The liver, in addition to dilatation of the biliary ducts, reveals atrophy

of the parenchymatous cells, focal necrosis, relative increase in the connective tissue, and in the event of chronic infection of the biliary ducts pericholangitic fibrosis, but no true cirrhosis; suppurative lesions may follow acute infections. Usually the growth does not invade adjacent tissues or organs, but it may grow into the pancreas, liver, or portal vein (and cause portal thrombosis). Metastases are uncommon, largely because of speedy death; they are most common in the liver (11 of 52 of Rolleston's cases), but they occur also in the regional lymphatic glands, the peritoneum (causing ascites), etc. Hemorrhage occasionally occurs; it may be due to so-called cholæmia, erosion of a vessel, or hemorrhagic pancreatitis.

Carcinoma of the ampulla of Vater (choledochopancreatic duct) should be distinguished from carcinoma of the termination of the common bile duct, of the termination of Wirsung's duct, and of the duodenal surface of the biliary capillaries. It begins, according to Rolleston, "as a thickening of the mucous membrane of the ampulla and infiltrates its muscular wall. It may form a villous or polypoid growth, and may then project through the orifice of the biliary papilla, which is dilated or ulcerated, into the duodenum." The growth tends to obstruct the orifice of Wirsung's duct and thus to produce dilatation of the intrapancreatic ducts and chronic interstitial pancreatitis; the same result may ensue upon extension to the orifice of Wirsung's duct of carcinoma of the lower end of the common bile duct. Secondary infection may result in suppurative cholangitis. Microscopically the growth is practically always a cylindrical-cell carcinoma (Rolleston).

Symptoms.—The onset of the symptoms referable to *carcinoma of the gall-bladder* may or may not be preceded by symptoms due to associated disease—cholecystitis and cholelithiasis. Some patients complain for years of what is commonly called dyspepsia, but which careful inquiry would show to be in reality chronic cholecystitis with perhaps pericholecystic adhesions; in other cases there is a more or less clear history of antecedent gallstones. There may have been many and recent attacks of obvious colic (with or without jaundice), or there may have been few, perhaps only one, remote and ill-defined attacks; that is, as perhaps occurs in most cases, the gallstones have remained latent, quiescent in the gall-bladder.

The onset of the symptoms referable to the carcinoma is insidious, and usually the disorder makes considerable progress before its true nature is suspected. The patient, usually an elderly woman, complains of ill-defined gastric symptoms, associated with more or less severe, sometimes colicky pain. These are usually ascribed to dietetic indiscretions; in some cases, however, the pain is so severe and perhaps referred to the right of the median line, and some local tenderness is found on examination, that the symptoms are put down to cholecystitis or cholelithiasis. The attack subsides, perhaps it recurs; the patient is thought to be the subject of cholelithiasis, and, indeed, in most cases, for some time the symptoms may be quite indistinguishable from cholelithiasis, or cholecystitis with pericholecystic adhesions.

Soon, however, the gall-bladder enlarges, jaundice supervenes, or the general health becomes impaired. An obvious palpable and often visible enlargement of the gall-bladder develops in more than one-half of the cases; in itself it is not significant of carcinoma, since it resembles that due to cholelithiasis or obstruction of the cystic duct; in advanced stages, however, it may become quite irregular and nodular, whereupon it is much more sig-

nificant of new-growth. Usually it reveals its attachment to the liver by moving with it during the phases of respiration and showing no intervening area of tympany; sometimes it is fixed to the abdominal wall by adhesions, and it may be quite tender. In some cases the tumor is the first obtrusive evidence of the disease, all symptoms being in abeyance until the enlarged gall-bladder discloses itself. Sooner or later, in most cases, the liver becomes involved (whereupon the symptoms become those of carcinoma of the liver), or the growth may extend to other adjacent tissues or organs or give rise to metastasis to the regional and other lymphatic glands.

Jaundice occurs in three-fourths or more of the cases (69 per cent., Musser; 86 per cent., Meurrier), but it is not always due to the same cause. Rarely it may result from duodenal catarrh or catarrhal cholangitis, last a short time, and subside; or it may be due to a gallstone in the common biliary duct, when it is likely to be intermittent and perhaps accompanied by intermittent fever; usually, however, it is due to extension of the new-growth into the biliary ducts, or to pressure from without on the extrahepatic biliary ducts by secondary deposits; the jaundice in this event is progressive, soon becoming very deep, and it is permanent. Upon the development of the jaundice the liver becomes uniformly enlarged, and presents a smooth surface, aside from the more local and irregular or nodular enlargement due to the new-growth.

With the progress of the disease the general health becomes impaired; this may be the first noteworthy evidence of its malignant nature. The patient gradually loses weight and strength, becomes anæmic, and presents the manifestations of cachexia. Dyspeptic symptoms develop, or become aggravated, in which event they may be due not only to the original cholecystitis or cholelithiasis, but also to an associated pyloric obstruction. Occasionally the carcinoma may invade the colon, giving rise to obstruction, or to a cholecystocolonic fistula, whence more or less serious infection of the gall-bladder may ensue. In other cases the gall-bladder forms adhesions with the peritoneum, and ultimately perhaps perforates thereinto, giving rise to a localized or more generalized peritonitis. Ascites occurs in about one-fourth of the cases, and is due most commonly to associated perihepatitis or peritonitis; rarely it may be due to obstruction of the portal circulation in consequence of extension of the original growth or of secondarily involved glands in the portal fissure. Edema of the legs is not uncommon in the late stages; usually it is due to cardiac debility, less commonly to pressure on or thrombosis of the inferior vena cava. Ultimately in the cases attended by jaundice the patient develops a condition attributed to hepatic insufficiency (hepatic toxæmia, cholæmia); he becomes drowsy and gradually passes into a semicomatose state; the breathing becomes superficial, the pulse slow, and the temperature perhaps elevated; submucous and subcutaneous hemorrhages occur; and in progressing asthenia, delirium, and coma the patient dies. In the absence of jaundice, the patient generally dies of asthenia. The disorder may run a fatal course within six months.

The symptoms of *carcinoma of the extrahepatic biliary ducts* are usually insidious. There may be antecedent dyspeptic symptoms of variable duration, and ill-defined complaints of disturbance of the general health. Jaundice is usually the first noteworthy symptom; in most cases it develops gradually; occasionally rather suddenly, imitating catarrhal cholangitis or a gallstone arrested in the common duct. The jaundice is progressive and

permanent; it soon becomes extreme, and biliary coloring matter disappears from the stools and appears in the urine. Pain in the right hypochondrium or the epigastrium is quite common, and is usually dull in character; sometimes it is severe, suggesting gallstone colic, but it is more likely due to spasmodic contraction of the gall-bladder against an obstruction. Obstruction to the flow of bile results in enlargement of the gall-bladder, which is palpable in one-half or more of the cases as a smooth pear-shaped swelling. The liver also is sometimes uniformly enlarged, but rarely notably so. Metastasis occurs in about one-half of the cases, but the nodules are usually small and not palpable during life—largely on account of the rapid course of the disease (five to six months). Should metastasis occur, it may lead to ascites. Infection of the biliary ducts and suppurative cholangitis may ensue. The general health soon becomes impaired; the patient loses strength and flesh, becomes anæmic and cachectic, and dies from exhaustion in delirium or coma. The symptoms of *carcinoma of the ampulla of Vater* are practically identical with those of carcinoma of the common bile duct.

Diagnosis.—Carcinoma of the gall-bladder is suggested by a history of ill-defined dyspeptic symptoms, perhaps interrupted by definite signs attributable to cholelithiasis or cholecystitis; pain in the right hypochondrium; jaundice; hard, nodular, and progressive enlargement of the gall-bladder; and gradual loss of flesh and strength. Attention to the sequence and grouping of the symptoms would probably permit of the diagnosis before the onset of jaundice, which is not a phenomenon of carcinoma of the gall-bladder itself, but of extension of the disease; but in the great majority of cases, jaundice supervenes before the patient is thought to be seriously ill. Carcinoma of the biliary ducts is suggested by insidious but persistent and progressive jaundice, smooth, uniform enlargement of the gall-bladder, loss of flesh and strength, in a patient about or over fifty years of age, without determinable cause of jaundice.

Carcinoma of the gall-bladder and biliary ducts is simulated by a number of other conditions, such as chronic cholecystitis and cholelithiasis, carcinoma and other tumors of adjacent organs. Chronic cholecystitis and cholelithiasis are, in most cases, antecedent to carcinoma of the gall-bladder, so that it is extremely difficult if not impossible, in most cases, to say when the new-growth begins. The diagnosis is favored by the detection of progressive nodular enlargement of the gall-bladder; but in many cases the carcinoma develops when the chronic inflammatory process has lasted a long time and caused much thickening and many adhesions to adjacent viscera, so that should the growth not attain at least moderate dimensions, no especially noteworthy enlargement of the gall-bladder becomes apparent. The diagnosis in these cases awaits the development of cachexia, or of metastasis, especially to the liver. Obviously then the diagnosis is often impossible until the disease has progressed so far as to be hopeless; sometimes it cannot be made with certainty even at operation.

In the early stages, carcinoma of the gall-bladder may sometimes be distinguished from carcinoma of the liver, by the absence of jaundice, due to limitation of the new-growth to the gall-bladder; but in the more advanced stages, when the disease is more widespread and involves the liver more or less extensively, the diagnosis may be quite impossible, although a history of antecedent cholelithiasis or cholecystitis, and the early development of a tumor in the region of the gall-bladder without jaundice, suggests that the

primary growth originated in the gall-bladder; while antecedent gastric, intestinal, genital, etc., symptoms, the presence of jaundice, and the early absence of a tumor in the region of the gall-bladder suggest that the liver rather than the gall-bladder was originally involved.

Carcinoma of the head of the pancreas, causing jaundice, can scarcely, if ever, be distinguished from carcinoma of the biliary ducts, aside from the fact that carcinoma of the head of the pancreas is much the more common, the pain is more likely to be epigastric than hypochondriac; a tumor deep in the epigastrium (close to the vertebrae) may sometimes be felt.

In the early stages, carcinoma of the gall-bladder and of the biliary ducts may sometimes be distinguished by the absence of jaundice and the presence of an enlarged and nodular gall-bladder, and the early presence of jaundice and a smooth and uniform enlargement of the gall-bladder. Later, when jaundice occurs in carcinoma of the gall-bladder, the jaundice being due to extension to the ducts or pressure by enlarged glands on the ducts, the differences are as slight anatomically as they are clinically.

Carcinoma of the ampulla of Vater closely resembles carcinoma of the biliary ducts. Rolleston states that the diagnosis may be regarded as very difficult or impossible, but that there are a few points of difference: in carcinoma of the ampulla (1) jaundice is said to be often intermittent, the faeces becoming bile-stained, and the icteric tint of the skin diminishing or even passing off in the earlier stages when the obstruction is possibly valvular, or partly due to spasm of the ducts set up by the irritation of the growth. Confusion is apt to arise between carcinoma of the ampulla and carcinoma of the duodenal surface of the papilla, in which jaundice is by no means constant; (2) intermittent hepatic fever and suppurative cholangitis are apt to occur; (3) diarrhoea is more often seen than in carcinoma of the ducts, in which constipation is the rule; attacks of diarrhoea may alternate with periods of obstinate constipation (Rolleston).

Prognosis.—Carcinoma of the gall-bladder and the biliary ducts is necessarily fatal, unless it can be removed by surgical intervention. Under non-surgical treatment patients seldom live more than six months from the onset of serious symptoms. The mortality is high also under surgical treatment, many patients succumbing to the operation itself, or to postoperative hemorrhage; in others operated upon late, the growth is too extensive to be removed; in others recurrences ensue several months after operation; or a fistula persists, and may lead to serious drains on the system and exhaustion.

Treatment.—The treatment is essentially surgical. Since there is no known means of cure at the hands of the physician, his energies should be directed toward making an early diagnosis and urging surgical intervention. Even the suspicion of malignant disease is an indication for operation; the hazard of the operation is less than the risks attendant upon delay. Since carcinomas occur in from 4 to 14 per cent. of cholelithitic subjects, and in a higher percentage of those with active manifestations, the operative treatment of cholelithiasis naturally tends to reduce the incidence of carcinoma of the gall-bladder; it is a prophylactic measure. Aside from the removal of the growth, a fistula may be established between the gall-bladder and the intestine in cases of carcinoma of the common duct or of the ampulla of Vater, and perhaps temporarily add to the patient's comfort. Aside from operation, the physician's efforts are limited to relieving distress and promoting the comfort of the patient.

INDEX.

A

ABDOMEN in acute enteritis, 374
 pancreatitis, 641, 642
 peritonitis, 522
 in anorexia nervosa, 164
 in appendicitis, 418
 in ascites, 504
 in cholera nostras, 375
 in chronic enteritis, 378
 peritonitis, 566
 in cirrhosis of stomach, 317
 in congenital hypertrophic stenosis
 of pylorus, 307
 in constipation, 358
 in diarrhoea, 369
 enlargement of, in idiopathic dilata-
 tion of colon, 364
 in enterocolitis, 385
 in intussusception, 461
 in mesenteric embolism, 489
 thrombosis, 489
 in mucous colitis, 492
 in nervous dyspepsia, 172
 in splachnoptosis, 607
 in subphrenic pneumothorax, 558
 in suppurative pancreatitis, 648
 in tuberculous of peritoneum, 572,
 573
 in tumors of peritoneum, 596
 in volvulus, 464
 Abdominal aorta, aneurism of, diagnosis
 of, from gastric carcinoma, 244
 muscles in duodenal ulcer, 391
 operations, acute dilatation of stoni-
 ach and, 281
 viscera, embryology of, 600
 wall, perforation of, in gastric ulcer,
 199
 walls, rigidity of, in acute diffuse
 peritonitis, 524
 Abscess, appendicular, subphrenic pneu-
 mothorax and, 557
 of appendix epiploica, 475
 of Fallopian tubes, subphrenic ab-
 scess and, 547
 of kidneys, subphrenic abscess and,
 547
 of liver, 745. *See* Liver, abscess of.
 of omentum, 542

Abscess of pancreas, 646. *See* Pancrea-
 titis, suppurative.
 peri-appendicular, 410, 421
 pericolic, 543.
 perigastric, 542.
 peritoneal, residual, 544
 retroperitoneal, 544.
 of spleen, subphrenic abscess and,
 546
 subphrenic, 545
 Absorption of foods by intestines, 331
 Acetic acid in carcinoma of stomach,
 237
 Acetone in carcinoma of stomach, 232
 Acholic stools, 335
 Achroöcytosis, 72
 Achylia gastrica, 29, 156
 in chronic gastritis, 273
 course of, 161
 definition of, 156
 diagnosis of, 160
 from carcinoma of stomach,
 161, 242
 etiology of, 157
 gastric contents in, 160
 neurasthenia and, 157
 pain in, 159
 pathology of, 158
 prognosis of, 161
 symptoms of, 159
 treatment of, 161
 vomiting in, 159
 Acid gastritis, diagnosis of, from hyper-
 acidity, 117
 Aene of mouth, 54
 pancreatica, 667
 Acoria, 162
 definition of, 162
 diagnosis of, 163
 from bulimia, 134, 163
 from polyphagia, 163
 etiology of, 162
 symptoms of, 162
 treatment of, 163
 Actinomycosis of appendix, 433
 of liver, 795
 of mouth, 60
 of oesophagus, 86
 Addison's disease, diagnosis of. from
 acute peritonitis, 534

- Addison's disease, diagnosis of, from carcinoma of stomach, 242
- Adenocarcinoma of stomach, 226
- Adenoma of intestines, 485
of liver, 801
of pancreas, 677
- Ærophagia, 341
- Aglossia, 61
- Agusia, 43
- Albuminuria in congestion of liver, 727
in carcinoma of stomach, 232
in cholera nostras, 376
in hæmatemesis, 320
in tuberculosis of peritoneum, 572
- Albumosuria in appendicitis, 418
- Alcohol, acute non-suppurative hepatitis and, 743
carcinoma of stomach and, 223
portal cirrhosis and, 760
- Alcoholism, acute yellow atrophy of liver and, 719
chronic interstitial pancreatitis and, 652
fatty liver and, 713
tuberculosis of peritoneum and, 568
- Allantois, cysts of, 590
- Allotriophagia, 135
- Aloin test for "occult bleeding," 194
- Amaurosis in hæmatemesis, 320
- Amœbæ in abscess of liver, 754
in carcinoma of stomach, 239
- Ampulla of Vater, carcinoma of, diagnosis of, 856
etiology of, 851
pathology of, 853
symptoms of, 855
treatment of, 856
- Amyloid degeneration, intestinal hemorrhage and, 350
of liver, 714. *See* Liver, amyloid, diagnosis of, from congestion, 734
disease of intestines, 439
diagnosis of, from tuberculosis, 437
tumors of tongue, 67
- Amylopsin, 330
- Anacidity, 156. *See* Achylia gastrica.
- Anadenia ventriculi, 271
- Anæmia in carcinoma of stomach, 232
in chronic dilatation of stomach, 290
enteritis, 379
diagnosis of, from portal cirrhosis, 782
in duodenal ulcer, 391
fatty liver and, 713
gastric ulcer and, 180, 188
treatment of, 214
intestinal hemorrhage and, 350
ulcers and, 398
in lead colic, 349
pernicious, diagnosis of, from carcinoma of stomach, 242
- Anæmia, splenic, diagnosis of, from portal cirrhosis, 781
portal cirrhosis and, 760
- Anæsthesia of œsophagus, 86
- Aneurism of abdominal aorta, diagnosis of, from carcinoma of stomach, 244
abdominal, diagnosis of, from acute peritonitis, 533
hæmoperitoneum and, 512
of hepatic artery, 702
ruptured, diagnosis of, from acute pancreatitis, 644
- Angina, Ludwig's, 75
pectoris, diagnosis of, from pneumatosis, 154
- Angiocholitis, 815
- Angioma of lips, 45
of liver, 801
of peritoneum, 582
- Angioneurotic œdema of mouth, 53
- Angiosarcoma, hæmoperitoneum and, 512
- Anomalies of biliary ducts, 802
of gall-bladder, 802
- Anorexia in chronic gastritis, 272
in cirrhosis of stomach, 316
in congenital pyloric stenosis, 306
nervosa, 163
abdomen in, 164
appetite in, 163
definition of, 163
diagnosis of, 164
etiology of, 163
inanition in, 164
symptoms of, 163
treatment of, 164
- Antiperistalsis, 328
pathological, 329
- Aorta, aneurism of, diagnosis of, from cysts of pancreas, 674
- Aphthous stomatitis, 46. *See* Stomatitis, aphthous.
- Apoplexy, pancreatic, 634
- Appendicitis, 400
abdominal distention in, 418
abscess of liver and, 747
abscesses and, 410, 421
age in, 404
albumosuria in, 418
bacteriology of, 407
catarrhal, 408, 420
chills in, 412, 416
cholelithiasis and, 828
chronic, 411, 421
localized peritonitis and, 560
relapsing, 422
treatment of, 432
constipation in, 412, 415
definition of, 400
diagnosis of, 422
abscess in, 423
of character and stage of, 423
differential, 424
from acute cholecystitis, 824

- Appendicitis, diagnosis of, from acute
 colitis, 425
 gastritis, 424
 intestinal obstruction, 425
 pancreatitis, 426
 peritonitis, 426, 427
 pyelitis, 427
 from biliary colic, 427
 from cholelithiasis, 427
 from cholera nostras, 376
 from diseases of gall-bladder,
 426
 from duodenal ulcer, 393, 426
 from gastric ulcer, 426
 from intestinal colic, 348, 424
 from malarial fever, 426
 from movable kidney, 427
 from nephrolithiasis, 427
 from nervous mimicry of ap-
 pendicitis, 428
 from oöphoritis, 428
 from ovarian neuralgia, 428
 from pneumonia, 426
 from salpingitis, 428
 from tuberculous peritonitis, 427
 from typhoid fever, 425
 digestive disturbances in, 405
 etiology of, 404
 local, 405
 faecal concretions and, 406
 fever in, 412, 415
 foreign bodies and, 406
 heredity in, 405
 hyperalgesia in, 413
 indicanuria in, 418
 leukocytosis in, 416
 limited to interior of appendix, 420
 lymphoid tissue of, influence of, 407
 nausea in, 412, 414
 nephropotosis and, 611
 occupation in, 404
 onset of, 412
 pain in, 412
 pathology of, 407
 peritonitis and, 410, 411, 418
 prognosis of, 428
 pulse in, 416
 pyæmia in, 419
 pyæmic abscess of liver and, 411
 race in, 404
 rigors in, 412
 septic thrombosis and, 411
 septicæmia in, 419
 sex in, 404
 signs of, 416
 subphrenic abscesses in, 419, 545
 symptoms of, 412
 reflex, 414
 treatment of, 430
 tumor in, 417
 urine in, 418
 varieties of, clinical, 419
 vomiting in, 415
- Appendicitis with involvement of sur-
 rounding structures, 421
 of whole thickness of organ, 420
 Appendicular colic, 346, 420
 Appendix, abscess of, subphrenic pneumo-
 thorax and, 557
 actinomycosis of, 433
 anatomy of, 401
 arteries of, 403
 carcinoma of, primary, 434
 secondary, 434
 circulation of, 403
 coats of, 402
 contents of, 409
 cystic distention of, 433
 diseases of, acute peritonitis and, 516
 empyema of, 409
 epiploica, abscess of, 475
 gangrene of, 410
 inflammation of, 400. *See* Appendi-
 citis.
 lymphatics of, 404
 mesentery of, 402
 mucocoele of, 433
 myoma of, 433
 myxoma of, 433
 necrosis of, 410
 nerves of, 404
 obliteration of, 412
 palpation of, 417
 polyps of, 433
 sarcoma of, 434
 shape of, 402
 size of, 402
 tuberculosis of, 432
 tumors of, 433
 valve of Gerlach, 403
 veins of, 404
 Aptyalism, 69
 Arterial spasm, gastric ulcer and, 183
 Arteries, mesenteric, embolism of, 487
 thrombosis of, 487
 Arteriosclerosis, chronic interstitial pan-
 creatitis and, 652
 perihepatitis and, 738, 741
 peritonitis and, 563
 gastric ulcer and, 180, 207
 of hepatic artery, 702
 of intestines, 490
 Artery, hepatic, diseases of, 701 *See*
 Hepatic artery, diseases of.
 Arthracotic cirrhosis of liver, 785
 Ascites, 498
 abdomen in, 504
 in acute perihepatitis, 736
 yellow atrophy of liver, 723
 backward pressure and, 499
 bacteriology of, 502
 in carcinoma of gall-bladder, 854
 of liver, 797
 of pancreas, 678, 680
 of stomach, 228
 cells in, 502, 508

- Ascites in chronic perihepatitis, 741
 peritonitis and, 498, 564, 566
 chyliform, 503
 chylous, 502
 cirrhosis of liver and, 499
 of stomach and, 317
 cysts and, 500
 of pancreas and, 672
 definition of, 498
 diagnosis of, 506
 as to cause, 507
 etiology of, 498
 exudates in, 501
 fatty, 503
 fluid in, 501, 508
 in hæmochromatosis, 783
 in hepatoptosis, 612
 hydatid cysts and, 500
 lactescent, 504
 lardaceous disease of liver and, 500
 in leukæmic infiltration of liver, 717
 milky, 504
 muscles in, 501, 505
 non-fatty, 504
 nutmeg liver and, 499
 pain in, 506
 palpation in, 505
 in passive congestion of liver, 733
 pathology of, 501
 percussion in, 505
 peritonitis and, 500
 in portal cirrhosis, 776
 fluid in, 782
 prognosis of, 509
 renal dropsy and, 500
 results of, 506
 signs of, 504
 skin in, 501, 505
 syphilis of liver and, 500
 thrombosis of portal vein and, 500, 707
 transudates in, 501
 treatment of, 509
 diuretics in, 511
 operative, 511
 paracentesis in, 509
 purgation in, 511
 tumors of liver and, 500
 veins in, 501, 505
- Asthma, dyspeptic, gastric atony and, 168
 Athleroma, mesenteric embolism and, 487
 Atonic dyspepsia, 279
 Atony, gastro-intestinal, hyperacidity and, 114, 116
 of intestines, gastric atony and, 168
 of stomach, 166
 Atrophic gastritis, 271
 Atrophy of liver, 710. *See* Liver, atrophy of.
 acute yellow, 718. *See* Liver, acute yellow atrophy of.
 of mucous membrane, diagnosis of, from carcinoma of stomach, 242
- Atrophy of pancreas, chronic interstitial pancreatitis and, 653
 of salivary glands, 72
 of tongue, smooth, 59
 Azotorrhœa, calculi of pancreas and, 661
 in diseases of pancreas, 624
- ### B
- BACILLUS *aërogenes* capsulatus in intestines, 337, 339
 botulinus in intestines, 339
 coli communis in appendicitis, 407
 calculi of pancreas and, 658
 in chronic peritonitis, 564
 in diarrhœa, 368
 in childhood, 382
 in fat necrosis, 630
 in intestinal obstruction, 448
 in intestines, 337, 339
 in mucous colitis, 491
 diphtheria in mouth, 40
 dysenteric in diarrhœa, 367, 368, 382
 enteritidis in diarrhœa, 368
 gangrenæ pulpæ, 40
 of glanders in mouth, 41
 lactis *aërogenes* in diarrhœa, 382
 in intestines, 337
 lepræ in mouth, 41
 necrodenialis, 40
 pseudodiphthericus in mouth, 40
 putrificus in intestines, 337, 339
 pyocyanus in intestines, 339
 in mouth, 40
 of rhinoscleroma in mouth, 41
 tuberculosis in peritonitis, 564
 in mouth, 40
- Bacteria, constipation and, 355
 in intestines, clinical results of, 339
 defensive role of, 338
 digestion and, 37
 distribution of, 336
 growth of, conditions influencing, 338
 significance of, 338
 treatment of, 340
 virulence of, conditions influencing, 338
- Bacteriology of acute cholecystitis, 817
 diffuse peritonitis, 517
 of appendicitis, 407
 of ascites, 501
 of diarrhœa, 368
 of intestinal tract, 336
 of subphrenic abscess, 552
 of suppurative pancreatitis, 647
 of tuberculosis of peritoneum, 569
- Bacterium gingivitis, 40
 Baelz's disease, 45
 Bands, intestinal obstruction due to, 454
 Bednar's aphthæ, 52

- Benzidin test for "occult bleeding," 194
 Bifid tongue, 61
 Bile duct, common, in cholelithiasis, 837
 secretion of, 32, 330
 Biliary cirrhosis, 790. *See* Liver, cirrhosis of, biliary.
 diagnosis of, from chronic catarrhal cholangitis, 816
 colic, in anomalies of liver, 100
 diagnosis of, from acute pancreatitis, 644
 from appendicitis, 427
 from gastric ulcer, 207
 from intestinal colic, 348
 ducts, anomalies of, 802
 signs of, 803
 carcinoma of, 850
 diagnosis of, 855
 etiology of, 851
 jaundice in, 854
 liver in, 852
 metastases in, 853, 855
 pathology of, 851
 prognosis of, 856
 symptoms of, 854
 treatment of, 856
 congenital obliteration of, 802
 sarcoma of, 850
 tumors of, suppurative cholangitis and, 812
 fistulae, 840
 tract, digestion and, 34
 infections of, 803
 bacteriology of, 804
 through diverticulum of Vater, 805
 lymphatic circulation, 806
 portal circulation, 805
 systemic circulation, 806
 wall of gall-bladder, 806
 treatment of, 845
 typhoid, 817
 inflammations of, 804
 cholelithiasis and, 827
 Bilirubin-calcium gallstones, 830, 831
 Black tongue, 62
 Bladder in appendicitis, 418
 distended, diagnosis of, from ascites, 507
 perforation into, in carcinoma of intestines, 484
 Bleeding tongue, 62
 Blood in abscess of liver, 754
 in acute diffuse peritonitis, 527
 yellow atrophy of liver, 723
 cultures in acute diffuse peritonitis, 528
 in diarrhoea in childhood, 386
 in jaundice, 694
 in portal cirrhosis, 779
 Blood in tuberculosis of peritoneum, 574
 Bloodvessels, diseases of, intestinal hemorrhage and, 350
 thrombosis of, in acute pancreatitis, 643
 Boas-Oppler bacilli in carcinoma of stomach, 238
 Boils in jaundice, 694
 Bowels, congestion of, constipation and, 355
 paralysis of, intestinal obstruction due to, 469
 strangulation of, intestinal hemorrhage and, 350
 ulceration of, 388
 Bradycardia in jaundice, 695
 Breath, offensive, 41
 in atony of stomach, 42
 in bronchitis, 42
 in carcinoma of larynx, 42
 in diabetes, 42
 in dilatation of stomach, 42
 in diverticulum of oesophagus, 42
 in hæmoptysis, 42
 in septicæmia, 42
 in uræmia, 42
 Bronchopneumonia in enterocolitis, 385
 Budd's non-alcoholic cirrhosis of liver, 760
 Bulimia, 133
 definition of, 133
 diagnosis of, 134
 from acoria, 134, 163
 from polyphagia, 134
 etiology of, 134
 prognosis of, 134
 symptoms of, 134
 treatment of, 134
 Burns, cutaneous, duodenal ulcer and, 393
 Butyric acid in carcinoma of stomach, 237
- ### C
- CACHECTIC splanchnoptosis, 608
 Cachexia in cirrhosis of stomach, 317
 fatty liver and, 713
 Calcareous infiltration of liver, 717
 Calculi of pancreas, 657
 salivary, 73
 Callous retroperitonitis, 310
 Cancer of stomach, 220. *See* Stomach, carcinoma of.
 Cancerum oris, 48
 Carbohydrates, absorption of, 331
 Carcinoma of appendix, 434
 of biliary ducts, 850
 cholelithiasis and, 828
 of colon, 481
 diagnosis of, from cholelithiasis, 842
 of duodenum, 479
 diagnosis of, from carcinoma of stomach, 243
 of gall-bladder, 850

- Carcinoma of ileum, 480
 of intestine, diagnosis of, from intestinal diverticula, 475
 of jejunum, 480
 of lips, 45
 of liver, 795
 diagnosis of, from carcinoma of stomach, 244
 of mouth, 52
 of œsophagus, 97
 of pancreas, 676
 calculi of pancreas and, 661
 diagnosis of, from suppurative pancreatitis, 650
 of peritoneum, 594
 of pylorus, diagnosis of, from duodenal ulcer, 393
 of stomach, 220
 developing upon gastric ulcer, 201
 diagnosis of, from achylia gastrica, 161
 from cirrhosis, 318
 from gastralgia, 137
 from gastric ulcer, 205
 from nervous dyspepsia, 173
 of tongue, 67
 Cardia in cirrhosis of stomach, 314
 spasm of, acute dilatation of stomach and, 282
 Cardiac cirrhosis, 731
 liver, 729
 stenosis, gastric ulcer and, 202
 Cardialgia in splachnoptosis, 607
 Cardiopospis, frequency of, 606
 Cardiospasm, 150
 definition of, 150
 diagnosis of, 151
 etiology of, 150
 symptoms of, 150
 treatment of, 151
 "Carriers," typhoid bacilli, 818
 Catarrhal appendicitis, 408, 420
 cholangitis, acute, 807
 chronic, 815
 cholecystitis, 819
 jaundice, 690, 807. *See* Cholangitis,
 catarrhal, acute.
 œsophagitis, 80
 stomatitis, 45
 ulcers of intestines, 395
 Cerebrospinal vomiting, 145
 Ceylon sore mouth, 441
 Chills in abscess of liver, 750, 751, 752
 in appendicitis, 412, 416
 in cholelithiasis, 832, 834
 in suppurative pancreatitis, 648
 pylephlebitis, 709
 Chloroform poisoning, acute yellow atrophy of liver and, 719
 Chlorosis, gastric ulcer and, 180
 hyperacidity and, 114
 Cholemia in carcinoma of gall-bladder, 854
 Cholangitis, catarrhal, 690
 acute, 807
 diagnosis of, 810
 from gallstones, 810
 from infectious jaundice, 810
 etiology of, 807
 faeces in, 809
 fever in, 809
 gall-bladder in, 809
 gastro-intestinal catarrh and, 807, 809
 jaundice in, 809
 liver in, 809
 pathology of, 808
 prognosis of, 810
 pulse in, 809
 symptoms of, 809
 treatment of, 810
 chronic, 815
 diagnosis of, 816
 from biliary cirrhosis, 816
 from gallstones, 816
 etiology of, 815
 gallstones and, 815
 pathology of, 815
 prognosis of, 816
 symptoms of, 816
 treatment of, 816
 in gastric ulcer, 199
 cholelithiasis and, 839
 radicular, 815
 suppurative, 811
 catarrhal cholangitis and, 812
 diagnosis of, 814
 from malarial fever, 815
 from suppurative pylephlebitis, 814
 etiology of, 811
 gall-bladder in, 813, 814
 gallstones and, 812
 jaundice in, 813
 leukoecytosis in, 814
 liver in, 813, 814
 organisms in, 811, 812
 pathology of, 812
 prognosis of, 815
 spleen in, 814
 symptoms of, 813
 treatment of, 815
 tumors of biliary ducts and, 812
 Cholecystitis, abscess of liver and, 746
 acute, 816
 bacteriology of, 817
 cystic duct in, 819
 diagnosis of, 823
 from appendicitis, 824
 etiology of, 816
 fever in, 821, 823
 fistulae in, 823

- Cholecystitis, acute, gall-bladder in, 819, 820
 gallstones and, 818
 jaundice in, 822, 823
 leukocytosis in, 822, 823
 pain in, 821
 pathology of, 819
 perforation in, 823
 peritonitis in, 823
 prognosis of, 824
 symptoms of, 820
 treatment of, 824
 typhoid bacillus in, 817, 818
 vomiting in, 821
- catarrhal, 819
- cholelithiasis and, 839
- chronic, 824
 cystic duct in, 824
 diagnosis of, 826
 from carcinoma of gall-bladder, 855
 etiology of, 824
 gall-bladder in, 824
 localized peritonitis and, 560
 pathology of, 824
 prognosis of, 826
 symptoms of, 825
 treatment of, 826
 ulcerative, 820
- gangrenous, 820
- membranous, 820
- phlegmonous, 820
- suppurative, 819
- Cholelithiasis, 826. *See also* Gallstones.
 acute cholecystitis and, 818
 pancreatitis and, 637, 643
 adhesions in, 839
 appendicitis and, 828
 associated lesions of, 832
 atony of stomach and, 167
 biliary fistula in, 840
 calculi of pancreas and, 657
 catarrhal cholangitis and, 815
 chills in, 832, 834
 cholangitis and, 839
 cholecystitis and, 839
 chronic interstitial pancreatitis and, 651
 common bile duct in, 837
 complications of, 839
 constipation and, 828
 crepitus of, 837
 cystic duct in, 837
 diagnosis of, 841
 from appendicitis, 427
 from carcinoma of gall-bladder, 855
 of pancreas, 681
 from catarrhal cholangitis, 810, 816
 from duodenal ulcer, 393, 842
 from gastralgia, 137
 from gastric ulcer, 842
- Cholelithiasis, diagnosis of, from hyper-acidity, 117
 x-rays in, 842
- diet and, 828
- diseases of lungs and, 828
- diverticulum of Vater in, 839
- etiology of, 826
- fever in, 832, 834, 837
- gall-bladder in, 836
- heart diseases and, 828
- hyperacidity and, 114
- inflammation of biliary tract and, 827
- intestinal obstruction in, 452, 841
- jaundice in, 834, 835
- metabolism and, 829
- mode of life and, 827
- pain in, 833, 834
- pancreas in, 839
- pathogenesis of, 831
- pathology of, 829
- peritonitis and, 839
- pregnancy and, 827
- prognosis of, 843
- prophylaxis of, 843
- puerperal infections and, 828
- pylephlebitis and, 839
- sequels of, 839
- sex in, 827
- splanchnoptosis and, 608
- suppurative cholangitis and, 812
- symptoms of, 832
 general, 833
- treatment of, 843
 diet in, 847
 medical, 847
 surgical, 848
 vomiting in, 833
- Choleperitoneum, 513
- Cholera bacillus in intestines, 339
- infantum, 385
- morbis, 385
- nostras, 375
 abdomen in, 375
 albuminuria in, 376
 collapse in, 376
 diagnosis of, 376
 from appendicitis, 376
 from typhoid fever, 376
 diarrhoea in, 375
 etiology of, 375
 fever in, 376
 onset of, 375
 pain in, 375
 pathological anatomy of, 375
 prognosis of, 376
 symptoms of, 375
 vomiting in, 375
- Choleraic diarrhoea in childhood, 385
- Cholesterin gallstones, 830, 831
- Chyliform ascites, 503
- Chylous ascites, 502
- Cicatrization in duodenal ulcers, 390
- Circulatory system, gastric ulcer and, 182

- Circumpapillary carcinoma, 480
- Cirrhosis of liver, 757. *See* Liver, cirrhosis of.
- Cirrhosis, biliary, 790. *See* Liver, cirrhosis of, biliary.
- cardiac, 731
- of haemochromatosis, 783
- hepatic anthracotic, 785
- of liver, diagnosis of, from chronic perihepatitis, 742
- from fatty liver, 714
- from portal thrombosis, 707
- from tuberculosis of peritoneum, 575
- malarial, 784
- portal, 759. *See* Liver, cirrhosis of, portal.
- of stomach, 271, 310. *See* Stomach, cirrhosis of.
- Cladobrix buccalis in mouth, 40
- Colic, biliary, in anomalies of liver, 700
- diagnosis of, from gastric ulcer, 207
- in calculi of pancreas, 660
- diagnosis of, from acute peritonitis, 532
- gallstone, 833
- intestinal, 345. *See* Intestinal colic.
- lead, 349. *See* Lead colic.
- nephritic, diagnosis of, from gastralgia, 137
- Colitis, 380
- acute, diagnosis of, from appendicitis, 425
- diagnosis of, 381
- diarrhoea in, 380
- etiology of, 380
- hemorrhage in, 380
- mucous, 490
- abdomen in, 492
- age in, 491
- bacillus coli communis in, 491
- complications of, 493
- constipation in, 492
- definition of, 490
- diarrhoea in, 492
- enteroptosis in, 493
- etiology of, 491
- hysteria and, 493
- intestinal sand in, 493
- membranous, 491
- mucous membrane in, 491
- neurasthenia in, 493
- pain in, 492
- pathological anatomy of, 491
- prognosis of, 493
- symptoms of, 492
- treatment of, 493
- ulceration in, 493
- pain in, 380
- pathological anatomy of, 380
- prognosis of, 381
- simple, 380
- Colitis, symptoms of, 380
- treatment of, 381
- ulcerative, 397
- Collapse in cholera nostras, 376
- in enterocolitis, 385
- in intestinal obstruction, 449
- in suppurative pancreatitis, 648
- Colloid cancer of peritoneum, 596
- Colon, carcinoma of, 481
- symptoms of, 481
- dilated, diagnosis of, from gastrectasis, 295
- dilatation of, idiopathic, 363
- abdomen in, 364
- constipation and, 356, 364
- diarrhoea in, 364
- etiology of, 363
- malnutrition in, 364
- symptoms of, 364
- treatment of, 365
- diseases of, acute peritonitis and, 516
- inflammation of, 380. *See* Colitis.
- transverse, perforation of, in acute pancreatitis, 643
- tumors of, diagnosis of, from carcinoma of stomach, 244
- ulcer of, 397
- subphrenic pneumothorax and, 557
- Coma in jaundice, 697
- in phlegmonous gastritis, 260
- Compression of bowel, 443, 458
- Concretions, intestinal obstruction and, 453
- Congenital anomalies of liver, 698
- hypertrophic stenosis of pylorus, 303
- jaundice, 802
- malformations of oesophagus, 79
- obliteration of biliary ducts, 802
- stenosis of oesophagus, 95
- Congestion of liver, 725
- passive, 729
- Constipation, 352
- abdomen in, 358
- in achylia gastrica, 160
- in acute diffuse peritonitis, 526
- pancreatitis, 641
- in appendicitis, 412, 415
- in atony of stomach, 167
- bacteria and, 355
- in carcinoma of colon, 481
- cholelithiasis and, 828
- in chronic dilatation of stomach, 289
- enteritis, 378
- gastritis, 272, 278
- localized peritonitis, 561
- in cirrhosis of stomach, 317
- complications of, 358
- in congenital stenosis of pylorus, 307
- congestion of bowels and, 355
- in continuous hypersecretion, 126
- definition of, 352
- diagnosis of, 358

- Constipation, diarrhoea in, 358
 dietetic causes of, 354
 diseases of stomach and, 354
 in duodenal ulcer, 391
 enteroptosis and, 356
 etiology of, 352
 functional, 354
 organic, 356
 faeces in, 358
 fever in, 357
 in gastric ulcer, 188
 in hyperacidity, 116
 idiopathic dilatation of colon and, 356, 364
 influence of dress on, 355
 of life on, 356
 in intestinal colic, 347
 diverticula, 475
 obstruction, 447
 in jaundice, 698
 in mucous colitis, 492
 in nervous dyspepsia, 172
 pain in, 357
 pathology of, 356
 pelvic tumors and, 356
 in splanchnoptosis, 607
 stenosis of intestines and, 356
 symptoms of, 357
 treatment of, 359
 diet, 360
 exercise, 360
 medical, 361
 surgical, 363
 uterine displacement and, 356
 in volvulus, 464
 of omentum, 469
 vomiting in, 357
- Constriction of intestines, 443
- Contractions of intestines, 329
- Convulsions in jaundice, 697
 in portal cirrhosis, 775
- Corrosive œsophagitis, 81
- Corset liver, 699
- Cruveilhier's disease, 175
- Crystals in faeces, 335
- Cynorexia, 133
- Cystic distention of appendix, 433
 duct in acute cholecystitis, 819
 carcinoma of, 850
 in cholelithiasis, 837
 in chronic cholecystitis, 824
- Cysticercus cellulosæ, 585
- Cysts, hydatid, chronic localized peritonitis and, 560
 of liver, 801
 of mesentery, diagnosis of, from cysts of pancreas, 673
 of mouth, 52
 ovarian, acute peritonitis and, 516
 chronic peritonitis and, 560
 diagnosis of, from cysts of pancreas, 673
 of pancreas, 663
- Cysts of pancreas, diagnosis of, from ovarian cysts, 673
 from suppurative pancreatitis, 650
 of parotid gland, 52
 of peritoneum, allantoic, 590
 conditions simulating, 590
 cysticercus cellulosæ, 585
 dermoid, 588
 hydatid, 583
 mesenteric, 585
 omental, 587
 retroperitoneal, 589
 sanguineous, 587
 urachal, 589
- Cytology in acute peritonitis, 521
- ### D
- DECUBITAL ulcers of intestines, 396
 of œsophagus, 84
- Delirium in hæmatemesis, 320
 in jaundice, 697
 in phlegmonous gastritis, 260
 in portal cirrhosis, 775
- Dermatoses of mouth, 53
- Dermoid cysts of peritoneum, 588
- Detoxifying function of liver, 686
- Diabetes in acute pancreatitis, 643
 breath in, 42
 bronzed, 783
 calculi of pancreas and, 660
 carcinoma of pancreas and, 677
 chronic interstitial pancreatitis and, 652, 655, 656
 cysts of pancreas and, 672
 pancreas in, 626
- Diacetic acid in carcinoma of stomach, 232
 in gastric ulcer, 202
- Diaphragm, perforation of, in gastric ulcer, 199
- Diaphragmatic hernia, diagnosis of, from subphrenic pneumothorax, 559
- Diarrhoea, 365
 in abscess of liver, 754
 in achylia gastrica, 160
 in amyloid disease of intestines, 440
 bacillus coli communis in, 368, 382
 dysenteric in, 367, 368, 382
 enteritidis in, 368
 in carcinoma of colon, 488
 of ileum, 480
 of jejunum, 480
 of pancreas, 679
 of stomach, treatment of, 248
 in childhood, 381
 age and, 383
 bacillus lactis ærogenes in, 382
 choleraic, 385
 dyspeptic, 384
 enterocolitis, 384
 abdomen in, 385

- Diarrhœa in childhood, enterocolitis, bronchopneumonia in, 385
collapse in, 385
diagnosis of, 385
diarrhœa in, 384
fever in, 384
œdema in, 385
prognosis of, 385
prolapse of rectum in, 385
urine in, 385
environment and, 383
etiology of, 381
food and, 381
inflammatory, 384
pathology of, 383
season and, 383
simple, 384
symptoms of, 384
treatment of, 386
in cholera nostras, 375
in chronic dilatation of stomach, 289
enteritis, 378
gastritis, 272
in colitis, 380
collapse in, 369
in congenital stenosis of pylorus, 307
definition of, 365
diagnosis of, 370
etiology of, 365
excessive ingestion of fluids and, 366
fever in, 369
food and, 366
hill, 442
Hippocratic facies in, 369
in hyperacidity, 116
in hypochlorhydria, 155
in idiopathic dilatation of colon, 364
in incontinence of pylorus, 166
in intestinal colic, 347
diverticula, 475
hemorrhage, 351
indigestion, 344
obstruction, 447
ulcers, 398
in intussusception, 461
in jaundice, 698
malarial fever and, 367
in mesenteric embolism, 489
thrombosis, 489
mucous, 491
colitis, 492
nervous influence and, 366
organic diseases of bowel and, 367
pneumonia and, 367
protozoa and, 368
purgative substances and, 366
renal diseases and, 367
in sarcoma of intestines, 485
septicopyæmia and, 367
staphylococci in, 368
streptococci in, 368
symptoms of, 368
- Diarrhœa, tenesmus in, 369
in toxic gastritis, 262
treatment of, 370
diet in, 370
medicinal, 370
in tuberculosis of intestines, 436
tubular, 491
in volvulus, 464
- Dietl's crises in nephropotosis, 610
- Digestion, adaptability in, 20
biliary tract and, 34
blood supply and, 32
chronic passive congestion and, 33
disharmony of secretion in, 34
disturbances of, functional, 21
general relations of, 17
individuality in disease of, 19
infection and, 32, 34, 38
interrelation of functions in, 19, 30, 31
intestinal flora and, 37
motor functions in, 21
peptic, inhibition of, 31
psychoneuroses and, 37
vicarious action in, 20
- Dilatation of colon, idiopathic, 363
in congenital stenosis of pylorus, 307
of hepatic artery, 701
veins, 703
of œsophagus, 90
in cardiospasm, 150
idiopathic, 85
of portal vein, 705
of stomach, acute, 280. *See* Stomach, dilatation of.
diagnosis of, from continuous hypersecretion, 127
- Diphtheria bacillus in mouth, 40
breath in, 42
- Diphtheritic gastritis, 267
- Diverticula, intestinal, 472
of œsophagus, 91
diagnosis of, from spasm, 88
pressure, 91
traction, 91, 92
- Divulsion in congenital hypertrophic stenosis of pylorus, 310
- Dry mouth, 69
- Duodenal hemorrhage, diagnosis of, from hæmatemesis, 322
stenosis, diagnosis of, 297
ulcer, 207, 389
acidity in, 209
age in, 207
anæmia in, 391
chronic peritonitis and, 560
cicatization and, 390
complications of, 210, 392
diagnosis of, 210, 392
from acute pancreatitis, 393
from appendicitis, 393, 426

- Duodenal ulcer, diagnosis of, from carcinoma of pylorus, 393
 from cholelithiasis, 393, 842
 from intestinal colic, 348
 obstruction, 393
 from portal cirrhosis, 781
 emaciation in, 391
 etiology of, 207, 389
 following cutaneous burns, 393
 hemorrhage in, 209, 390, 391
 hyperacidity in, 391
 intestinal hemorrhage and, 350
 number of, 209
 pain in, 209, 390
 pathological anatomy of, 389
 perforation in, 210, 390, 391
 peritonitis in, 392
 secondary changes in, 390
 sex in, 208
 site of, 208
 stenosis of duodenum in, 392
 subphrenic abscess and, 547
 pneumothorax and, 556
 symptoms of, 209, 390
 treatment of, 393
 vomiting in, 209, 391
- Duodenitis, acute pancreatitis and, 635
- Duodenum, carcinoma of, 479
 of pancreas and, 678
 circumpapillary, 480
 diagnosis of, from carcinoma of ampulla of Vater, 480
 of pancreas, 480
 of stomach, 243
 intrapapillary, 480
 suprapapillary, 479
 diagnosis of, from cancer of pylorus, 479
 symptoms of, 479
 perforation of, in acute pancreatitis, 643
 in suppurative pancreatitis, 649
 stenosis of, in duodenal ulcer, 392
 traction of, acute dilatation of stomach and, 281
- Dysentery, abscess of liver and, 746
 acute non-suppurative hepatitis and, 743
 bacillus in intestines, 339
 diagnosis of, from colitis, 381
- Dysphagia in acute oesophagitis, 81
 in carcinoma of oesophagus, 100
 in cardiospasm, 150
 in spasm of oesophagus, 88
 in toxic gastritis, 262
- Dyspepsia, atonic, 279
 fermentative, stools in, 344
 in gastric ulcer, 188
 nervous, 170
 definition of, 170
 diagnosis of, 172
 from chronic gastritis, 173
- Dyspepsia, nervous, diagnosis of, from gastrectasis, 295
 from gastric ulcer, 173
 etiology of, 171
 gastric atony and, 172
 gastroptosis and, 172
 palpitation of heart in, 172
 pathology of, 171
 symptoms of, 171
- Dyspeptic asthma, gastric atony and, 168
 cirrhosis of liver, 760
 diarrhoea in childhood, 384
- Dyspnœa in acute dilatation of stomach, 283
 in carcinoma of oesophagus, 101
 in cardiospasm, 150
 in dilatation of oesophagus, 90
 in passive congestion of liver, 734
 in spasm of oesophagus, 88
- E**
- ECHINOCOCCUS cysts, diagnosis of, from cysts of pancreas, 673
 from tumors of peritoneum, 597
- Ectasia of oesophagus, 90
- Ectopic gestation, hæmoperitoneum and, 511
 rupture of, diagnosis of, from acute peritonitis, 533
- Eczema of mouth, 54
- Effusion in tuberculosis of peritoneum, 572
- Emaciation in chronic dilatation of stomach, 289
 enteritis, 378
 in duodenal ulcer, 391
 in gastrosuccorrhœa, 129
 in mucous colitis, 492
 in suppurative pancreatitis, 649
- Embolie ulcer of intestines, 395
- Embolism, gastric ulcer and, 182
 of hepatic artery, 701
 veins, 703
 of mesenteric arteries, 487
 of portal vein, 708
 pulmonary, in subphrenic abscess, 553
- Emphysema, diagnosis of, from subphrenic abscess, 554
 in subphrenic pneumothorax, 558
- Emphysematous liver, 712
- Empyema of appendix, 409
 subphrenic abscess and, 547
- Endocarditis, embolism of mesenteric arteries and, 487
 gastric ulcer and, 180
- Endophlebitis of hepatic veins, 705
 of portal vein, 710
- Endothelioma of peritoneum, 591
- Enteritis, acute, 372
 definition of, 372

- Enteritis, acute, etiology of, 372
 faces in, 373
 pain in, 374
 pathology of, 372
 symptoms of, 373
 treatment of, 377
 urine in, 375
 vomiting in, 374
 chronic, 378
 diagnosis of, 379
 diarrhoea in, 378
 emaciation in, 378
 etiology of, 378
 pathology of, 378
 prognosis of, 379
 symptoms of, 378
 treatment of, 379
 Enterocolitis, acute, diagnosis of, from
 acute peritonitis, 532
 in childhood, 384
 diagnosis of, from colitis, 381
 from intestinal obstruction, 451
 Enterokinase, 331
 Enteroptosis, 599. *See* Splanchnoptosis.
 Epidemic gangrenous proctitis, 442
 Epiploitis, 541
 definition of, 541
 symptoms of, 542
 treatment of, 542
 Epithelial cells in mucous colitis, 491
 Erepsin, 331
 Eructation, nervous, 143
 definition of, 143
 diagnosis of, 144
 etiology of, 143
 symptoms of, 143
 treatment of, 144
 Erysipelas of mouth, 54
 Erythema in chronic dilatation of stom-
 ach, 289
 Erythrocytes in jaundice, 695
 Exfoliative cesophagitis, 80
 Extraperitoneal subphrenic abscess, 551
 Eye-strain, vomiting and, 147

F

- FACIES in abscess of liver, 753
 in acute peritonitis, 523
 in tuberculosis of peritoneum, 573
 Faecal concretions, appendicitis and, 406
 masses, intestinal obstruction due to,
 452
 Faeces, 333
 in acute catarrhal cholangitis, 809
 enteritis, 373
 pancreatitis, 642
 yellow atrophy of liver, 722
 in amyloid disease of intestines, 440
 in carcinoma of intestines, 483
 of pancreas, 679
 chemical composition of, 334
 Faeces in circumpapillary carcinoma, 480
 color of, 333
 consistency of, 333
 in constipation, 358
 crystals in, 335
 in diseases of pancreas, 622, 624
 inorganic constituents of, 334
 in intestinal hemorrhage, 350
 indigestion, 344
 in jaundice, 698
 in lymphosarcoma of intestines, 485
 microscopic examination of, 334
 odor of, 333
 reaction of, 334
 in sarcoma of intestines, 485
 in sprue, 441
 in tuberculosis of intestines, 436
 of peritoneum, 574
 Fallopian tubes, abscess of, subphrenic
 abscess and, 547
 tuberculosis of, tuberculosis of
 peritoneum and, 569
 Fat necrosis, 629
 bacillus coli in, 630
 clinical significance of, 632
 definition of, 629
 etiology of, 629
 experimental, 630
 incidence of, 630
 pancreatitis and, 630, 631
 pathological anatomy of, 631
 urine in, 633
 Fats, absorption of, by intestines, 332
 Fatty acids, portal cirrhosis and, 760
 ascites, 503
 liver, 712. *See* Liver, fatty.
 stools, 335
 Ferments in carcinoma of stomach, 238
 in cysts of pancreas, 670
 Fever in abscess of liver, 750, 751, 752
 in acute catarrhal cholangitis, 809
 cholecystitis, 821, 823
 enteritis, 374
 non-suppurative hepatitis, 745
 pancreatitis, 641, 642
 perihepatitis, 736
 peritonitis, 527
 in appendicitis, 412, 415
 in cholelithiasis, 832, 834, 837
 in cholera nostras, 376
 in colitis, 380
 in constipation, 357
 in diarrhoea, 369
 in childhood, 386
 in enterocolitis, 384
 in hæmatemesis, 320
 in intestinal ulcers, 398
 in phlegmonous gastritis, 260
 in portal cirrhosis, 773, 775
 in subphrenic abscess, 553
 pneumothorax, 558
 in suppurative pancreatitis, 648
 in tuberculosis of intestines, 436

- Fever in tuberculosis of peritoneum, 574
 Fibroma of liver, 801
 of œsophagus, 103
 of peritoneum, 579
 of tongue, 67
 Fibromyoma of œsophagus, 104
 of peritoneum, 580
 Fibropurulent peritonitis, progressive, 520
 Fibrous stenosis of œsophagus, 95
 Fistula, gastrocolic, in carcinoma of intestines, 483
 Fistulæ in acute cholecystitis, 823
 Flagellata in carcinoma of stomach, 239
 Follicular œsophagitis, 80
 ulcers of intestines, 396
 Food, absorption of, by intestines, 331
 diarrhœa in childhood and, 381
 Foreign bodies, appendicitis and, 406
 intestinal obstruction due to, 452, 453
 pericolic abscesses and, 544
 Functional digestive disturbances, 21
 organic disease and, 23
 structural change and, 24
 paralysis, intestinal obstruction and, 469
 Fürbringer's sign in subphrenic abscess, 555
- G**
- GALL-BLADDER in acute cholangitis, 809
 cholecystitis, 819, 820
 anomalies of, 802
 carcinoma of, 850
 ascites in, 854
 diagnosis of, 855
 from carcinoma of liver, 855
 of pancreas, 856
 from cholecystitis, 855
 from cholelithiasis, 855
 etiology of, 851
 jaundice in, 853
 pathology of, 851
 prognosis of, 856
 symptoms of, 853
 treatment of, 856
 in carcinoma of liver, 798, 852
 of pancreas, 680
 in cholelithiasis, 836
 in chronic cholecystitis, 824
 perihepatitis, 739
 diseases of, diagnosis of, from appendicitis, 426
 distended, diagnosis of, from ascites, 507
 from cysts of pancreas, 673
 from nephroptosis, 615
 perforation of, diagnosis of, from acute pancreatitis, 644
 in gastric ulcer, 199
 sarcoma of, 850
 in suppurative cholangitis, 813, 814
- Gall-bladder, tumors of, diagnosis of, from carcinoma of stomach, 244
 Gallstones, 826. *See also* Cholelithiasis.
 acute cholecystitis and, 818
 chronic catarrhal cholangitis and, 815
 colic, 833
 treatment of, 846
 crepitus of, 837
 diagnosis of, from acute catarrhal cholangitis, 810
 from chronic cholangitis, 816
 from duodenal ulcer, 393
 from gastralgia, 137
 discharge of, 845
 intestinal obstruction and, 452
 solution of, 844
 splachnoptosis and, 608
 suppurative cholangitis and, 812
 varieties of, 830
 Gangrene of appendix, 410
 of kidney in nephroptosis, 610
 Gangrenous cholecystitis, 820
 pancreatitis, 638
 fat necrosis and, 630
 stomatitis, 48
 Gaseous liver, 712
 Gastralgia, diagnosis of, from gastric ulcer, 205
 nervous, 135
 diagnosis of, 137
 etiology of, 136
 pain in, 136
 symptoms of, 136
 treatment of, 137
 vomiting in, 136
 Gastralgokenosis, 135
 Gastrectasis, 278. *See also* Stomach, dilatation of.
 diagnosis of, from gastric atony, 168
 gastric secretion and, 286
 gastroptosis and, 287
 idiopathic, 285
 movable kidney and, 287
 obstructive, 288. *See* Stomach, dilatation of.
 relative, 288
 symptomatic, 288
 Gastrectomy in carcinoma of stomach, 251
 Gastric contents in gastrectasis, 293
 crises, 145
 diagnosis of, from gastrectasis, 295
 disease, carcinoma of stomach and, 222
 disturbances in idiopathic dilatation of colon, 364
 emptiness, painful, 135
 hyperæsthesia, 138
 definition of, 138
 diagnosis of, 139
 etiology of, 138
 symptoms of, 138
 treatment of, 139

- Gastric idiosyncrasies, 139
 juice, hypersecretion of, 122. *See*
 Gastrosuccorrhœa.
 neuroses, 105
 age in, 107
 classification of, 108
 definition of, 105
 diagnosis of, 109
 from chronic gastritis, 274
 etiology of, 106
 occurrence of, 107
 prognosis of, 110
 summary of, 174
 treatment of, 110
 regurgitation, 141
 definition of, 141
 diagnosis of, 142
 from cesophageal regurgita-
 tion, 142
 from vomiting, 142
 etiology of, 142
 prognosis of, 142
 symptoms of, 142
 treatment of, 142
 secretion, gastrectasis and, 286
 ulcer, 175. *See also* Stomach, ulcer of.
 age in, 177
 anæmia in, 180, 188
 treatment of, 214
 arterial spasm and, 183
 arteriosclerosis and, 180
 carcinoma developing upon, 201
 diagnosis of, 201
 symptoms of, 201
 treatment of, 202
 of stomach and, 222
 cardiac stenosis and, 202
 chlorosis and, 180
 chronic peritonitis and, 560
 circulatory system and, 182
 complications of, 196
 constipation in, 188
 course of, 210
 definition of, 175
 diaceturia and, 202
 diagnosis of, 204
 from appendicitis, 426
 from arteriosclerosis, 207
 from biliary colic, 207
 from carcinoma, 205
 from cirrhosis of stomach,
 318
 from gastralgia, 205
 from gastritis, 205
 from gastropotosis, 615
 from hemorrhagic erosion
 with exfoliation, 206
 oozings from mucous
 membrane, 206
 from hyperæsthesia, 205
 from hyperchlorhydria, 205
 from hysteria with vomit-
 ing, 205
- Gastric ulcer, diagnosis of, from intes-
 tinal colic, 348
 from pregnancy with hy-
 peremesis, 205
 from spinal disease, 207
 from uræmia, 205
 diet and, 180
 diminished alkalinity of blood
 and, 183
 duration of, 210
 dyspepsia in, 188
 emaciation in, 188
 embolism and, 182
 endocarditis and, 180
 etiology of, 26, 177
 fever in, 188
 functions and, 26
 gastric analysis in, 195
 gastroduodenostomy in, 218
 gastro-enterostomy in, 218, 220
 geographical distribution of, 176
 hæmatemesis in, 190
 signs of, 191
 symptoms of, 191
 treatment of, 214
 surgical, 219
 healing of, 187
 hemorrhage in, operation for,
 218, 219
 hemorrhages, submucous, and,
 182
 hemorrhagic erosions and, 182
 histology of, 187
 history of, 175
 hour-glass stomach and, 202
 etiology of, 203
 symptoms of, 203
 treatment of, 220
 hyperacidity and, 182
 infections and, 180
 intestinal hemorrhage and, 350
 latent, 187
 leukocytosis in, 200
 morbid anatomy of, 185
 neuropathic theory of, 184
 number of, 185
 objective signs of, 195
 "occult bleeding" in, 192
 occupation in, 179
 pain in, 188
 cause of, 189
 effect of food on, 189
 time of, 189
 treatment of, 214
 parotitis and, 202
 pathogenesis of, 181
 perforation in, 197
 into abdominal wall, 199
 course of, 199
 into diaphragm, 199
 into gall-bladder, 199
 into intestines, 199
 into liver, 199

- Gastric ulcer, perforation in, into pan-
 creas, 199
 into pericardium, 199
 into pleura, 199
 into portal vein, 199
 into spleen, 199
 pain in, 200
 signs of, 199
 symptoms of, 199
 treatment of, surgical, 219
 perigastritis in, 196
 precardiæ, 197
 pyloric, 197
 symptoms of, 197
 perisplenitis and, 199
 peritonitis and, 198
 phlegmonous gastritis and, 198
 prognosis of, 210
 pseudoperforation of, 200
 pyelephlebitis and, 199
 pyloric stenosis and, 202
 pyloroplasty, 218
 pyopneumothorax in, 199
 pyrosis in, 188
 sequelæ of, 196
 sex in, 178
 shape of, 185
 situation of, 185
 size of, 187
 statistics of, 176
 subphrenic abscess and, 198, 546
 pneumothorax and, 556
 symptoms of, 187
 general, 188
 special, 188
 syphilis and, 180
 tetany and, 202
 thrombosis and, 182
 trauma in, 179
 treatment of, 211
 Cohnheim's, 216
 diet in, 212
 Lenhart's, 214
 Leube's, 214
 medicinal, 213
 mouth feeding in, 212
 prophylactic, 211
 Senator's, 215
 surgical, 216
 indications for, 218
 methods of, 218
 trichiniasis and, 181
 tuberculosis and, 181
 tumor in, 195
 vomiting in, 190
 treatment of, 214
- Gastritis, acid, diagnosis of, from hyper-
 acidity, 117
 acute, 253
 classification of, 253
 definition of, 253
 diagnosis of, from appendicitis,
 424
- Gastritis, acute, hemorrhagic pancreatitis
 and, 635
 simple, 253
 course of, 257
 definition of, 253
 diagnosis of, 256
 diarrhœa in, treatment of,
 258
 etiology of, 254
 chemical, 254
 mechanical, 254
 parasitic, 255
 animal, 255
 vegetable, 255
 thermal, 254
 nausea in, treatment of, 258
 pain in, 256
 pathology of, 255
 primary, 254
 prognosis of, 257
 secondary, 255
 signs of, 256
 symptoms of, 255
 treatment of, 257
 vomiting in, 256
 treatment of, 258
- atrophic, 271
 chronic, 257
 achylia in, 273
 anorexia in, 272
 atrophic, 270
 bowels in, 272
 classification of, 268
 definition of, 267
 diagnosis of, 274
 from achylia gastrica, 275
 from cancer of stomach, 275
 from carcinoma, 241
 from gastralgia, 137
 from gastrectasis, 295
 from gastric hyperæsthesia,
 139
 neuroses, 274
 ulcer, 205
 from nervous dyspepsia, 173
 etiology of, 269
 gastrectasis in, 273
 gastric contents in, 272
 hyperacidity in, 273
 incidence of, 267
 morbid anatomy of, 270
 neurasthenia in, 272
 physical examination in, 272
 productive, 270
 prognosis of, 274
 submucous sclerosis with, 310
 symptoms of, 272
 treatment of, 275
 vomiting in, 272
- cystica, 271
 diphtheritic, 265
 definition of, 265
 etiology of, 266

- Gastritis, diphtheritic, morbid anatomy of, 266
 symptoms of, 267
 granulosa, 270
 hypertrophic stenosing, 310
 interstitial, chronic, 310
 purulent, 258
 membranacea, 265
 mycotic, 263
 etiology of, 264
 symptoms of, 265
 treatment of, 265
 phlegmonous, 258
 age in, 259
 circumscribed, 259
 coma in, 260
 course of, 260
 definition of, 258
 delirium in, 260
 diagnosis of, 260
 from perigastric abscess, 543
 diffuse, 259
 etiology of, 258
 fever in, 260
 in gastric ulcer, 198
 leukocytosis in, 261
 morbid anatomy of, 259
 perigastritis and, 259
 prognosis of, 261
 sex in, 259
 symptoms of, 260
 treatment of, 261
 polyposa, 270
 submucosa, 258
 toxic, severe, 261
 diagnosis of, 263
 diarrhœa in, 262
 dysphagia in, 262
 etiology of, 261
 jaundice in, 263
 nausea in, 262
 onset of, 262
 pain in, 262
 petechiæ in, 263
 prognosis of, 263
 symptoms of, 262
 treatment of, 263
 vomiting in, 262
 Gastrocolic fistula in carcinoma of intestines, 483
 Gastroduodenitis, acute pancreatitis and, 636
 Gastroduodenostomy in gastric ulcer, 218
 Gastro-enterostomy in carcinoma of stomach, 252
 in congenital hypertrophic stenosis of pylorus, 310
 in gastrectasis, 303
 in gastric ulcer, 218, 220
 Gastrohemorrhagia, 319
 Gastro-intestinal atony, hyperacidity and, 114, 116
 splanchnoptosis, 608
 Gastromyxorrhœa, 130
 continuous, 132
 definition of, 130
 gastric contents in, 131
 intermittent, 132
 Gastroplication in gastrectasis, 303
 Gastropotosis, 608
 diagnosis of, 613
 from carcinoma of stomach, 615
 from gastrectasis, 295
 from gastric ulcer, 615
 frequency of, 605
 gastrectasis and, 287
 nervous dyspepsia and, 172
 symptoms of, 609
 treatment of, operative, 619
 Gastrostomy in carcinoma of stomach, 253
 Gastrosuccorrhœa, 122
 chronic, in pylorospasm, 153
 continua chronica, 125
 atony of stomach in, 127
 constipation in, 126
 definition of, 125
 diagnosis of, 127
 dilatation of stomach in, 127
 etiology of, 125
 pathology of, 126
 prognosis of, 127
 sequelæ of, 127
 symptoms of, 126
 treatment of, 125
 vomiting in, 126
 periodica, 122
 definition of, 122
 diagnosis of, 124
 etiology of, 122
 prognosis of, 124
 symptoms of, 123
 treatment of, 124
 vomiting in, 123
 definition of, 122
 diagnosis of, from dilatation of stomach, 127
 digestive, 128
 definition of, 128
 diagnosis of, 129
 etiology of, 128
 gastric contents in, 129
 prognosis of, 130
 symptoms of, 129
 treatment of, 130
 Geographical tongue, 63
 Gerlach, valve of, 403
 Glanders, bacillus of, in mouth, 41
 Glénard's disease, 599. *See* Splanchnoptosis.
 Globus hystericus in nervous dyspepsia, 172
 Glossitis, acute, 63
 chronic superficial, 64
 Moeller's, 64
 papillaris, 63

Glossodynia, 64
 Glycogenic function of liver, 687
 Glycosuria in acute pancreatitis, 641, 642
 alimentary, calculi of pancreas and, 661
 diseases of pancreas and, 628
 in carcinoma of pancreas, 680
 in chronic pancreatitis, 655
 diseases of pancreas and, 626
 in suppurative pancreatitis, 649
 Gonococcal infection, chronic localized peritonitis and, 560
 peritonitis, 530
 stomatitis, 51
 Gums, tuberculosis of, 58
 Gustatory paresthesia, 43

H

HÆMANGIOMA of lips, 45
 Hæmatemeses, 319
 in acute yellow atrophy of liver, 722
 albuminuria in, 320
 amaurosis in, 320
 in carcinoma of stomach, 230
 in cirrhosis of stomach, 316
 definition of, 319
 delirium in, 320
 diagnosis of, 321
 from carcinoma of stomach, 324
 from duodenal hemorrhage, 322
 from hemorrhagic erosions, 323
 from miliary aneurisms, 323
 from œsophageal varices, 322
 from ulcer of stomach, 323
 from varices in stomach, 323
 from venous stasis, 323
 from vicarious hemorrhages, 323
 etiology of, 319
 extragastric, 320
 fever in, 320
 gastric, 319
 in gastric ulcer, 190
 treatment of, 214, 218, 219
 leukocytosis in, 320
 morbid anatomy of, 319
 neuropathic, diagnosis of, 322
 optic neuritis in, 320
 pain in, 320
 in portal cirrhosis, 773
 prognosis of, 324
 in pyelephlebitis, 709
 symptoms of, 319
 subjective, 320
 in thrombosis of portal vein, 707
 treatment of, 325
 accessory, 326
 surgical, 326
 Hæmatoma, retroperitoneal, 588
 Hæmaturia in nephropotosis, 610
 Hæmochromatosis, 654
 cirrhosis of liver in, 783

Hæmoperitoneum, cirrhosis of liver in,
 diagnosis of, 784
 etiology of, 783
 pancreas in, 783
 prognosis of, 784
 spleen in, 783
 symptoms of, 783
 treatment of, 784
 Hæmoperitoneum, 511
 aneurism and, 512
 angiosarcoma and, 512
 diagnosis of, 512
 ectopic gestation and, 511
 etiology of, 511
 hemorrhage into suprarenal bodies and, 512
 pain in, 512
 prognosis of, 512
 rupture of liver and, 511
 of mesentery and, 511
 of spleen and, 511
 sequelæ of, 512
 symptoms of, 512
 treatment of, 513
 Hæmoptysis, breath in, 42
 Hanot's cirrhosis of liver, 758
 Harelip, 45
 Headache in acute peritonitis, 528
 in constipation, 357
 in idiopathic dilatation of colon, 364
 Heart diseases, cholelithiasis and, 828
 palpitation of, in nervous dyspepsia, 172
 portal cirrhosis and, 778
 valvular disease of, intestinal hemorrhage and, 350
 Heartburn in continuous hypersecretion, 126
 Helminthiasis, salivation in, 69
 Hemangioma of intestines, 486
 Hemeralopia in jaundice, 697
 Hemorrhage from tongue, 62
 in acute yellow atrophy of liver, 721, 722
 in aneurism of hepatic artery, 702
 in benign tumors of intestines, 486
 in carcinoma of ileum, 480
 of jejunum, 480
 in colitis, 380
 in duodenal ulcer, 209, 390, 391
 intestinal, 349, 398. *See* Intestinal hemorrhage.
 in intussusception, 462
 in mesenteric embolism, 489
 thrombosis, 489
 into pancreas, 634
 submucous, gastric ulcer and, 182
 vicarious, diagnosis of, from hæmatemeses, 323
 Hemorrhagic cysts of pancreas, 669
 erosion, diagnosis of, from gastric ulcer, 206
 from hæmatemeses, 323

- Hemorrhagic erosion, gastric ulcer and, 182
 oozing, diagnosis of, from gastric ulcer, 206
 pancreatitis, acute, 633. *See* Pancreatitis, acute hemorrhagic.
- Hemorrhoids, intestinal hemorrhage and, 350
 in mucous colitis, 491
 in portal cirrhosis, 773
- Hepatic abscess, diagnosis of, from ascites, 507
 artery, aneurism of, 702
 diagnosis of, 703
 pathology of, 702
 symptoms of, 702
 treatment of, 703
 arteriosclerosis of, 702
 dilatation of, 701
 diseases of, 701
 embolism of, 701
 thrombosis of, 701
 splanchnoptosis, 608
 veins, dilatation of, 703
 diseases of, 703
 embolism of, 703
 endophlebitis of, 704
 suppurative phlebitis of, 704
 thrombophlebitis of, 704
 thrombosis of, 703
- Hepatitis, acute, 743
 non-suppurative, 743
 alcohol and, 743
 diagnosis of, 745
 from acute yellow atrophy, 745
 from phosphorus poisoning, 745
 from suppurative hepatitis, 745
 dysentery and, 743
 etiology of, 743
 fever in, 745
 jaundice in, 744
 liver in, 744
 pain in, 744
 pathology of, 743
 prognosis of, 745
 spleen in, 744
 symptoms of, 744
 treatment of, 745
 parenchymatous, 743
 chronic, interstitial, 757
 suppurative, 745. *See* Liver, abscess of.
- Hepatomphalos, 698
- Hepatoptosis, 612, 701
 ascites in, 612
 diagnosis of, 614
 from hydronephrosis, 615
 from nephroptosis, 615
 from renal tumors, 615
 frequency of, 606
- Hepatoptosis, hepatopexy in, 620
 symptoms of, 612
 treatment of, operative, 620
- Hepatopexy in hepatoptosis, 620
- Hernia, diaphragmatic, diagnosis of, from subphrenic pneumothorax, 559
 intestinal hemorrhage and, 350
 obstruction due to, 455
- Herpes buccalis, 51
 labialis, 44
 of cesophagus, 89
- Herpetie stomatitis, 51
- Heterochylia, 156
- Hiccough in acute perihepatitis, 736
 peritonitis, 526
- Hill diarrhœa, 442
- Hippoeratic facies in diarrhœa, 369
 fingers in biliary cirrhosis, 793
- Hormones, gastric secretion and, 29
- Hour-glass stomach, 202. *See* Stomach, hour-glass.
- Hutchinsonian teeth, 59
- Hyaline perihepatitis, 737
- Hyaloserositis, 737, 740
- Hydatid cysts, chronic localized peritonitis and, 560
 of liver, subphrenic pneumothorax and, 556
 of peritoneum, 583
- Hydronephrosis, carcinoma of pancreas and, 678
 diagnosis of, from ascites, 507
 from cysts of pancreas, 674
 from hepatoptosis, 615
 in nephroptosis, 610
- Hyperacidity, 111
 chlorosis and, 114
 cholelithiasis and, 114
 in chronic gastritis, 273
 constipation in, 116
 definition of, 111
 diagnosis of, 117
 from acid gastritis, 117
 from atony of stomach, 117
 from cholelithiasis, 117
 from chronic hypersecretion, 117
 from ulcer of stomach, 117
 diarrhœa in, 116
 diet and, 114
 duodenal ulcer and, 391
 etiology of, 113
 gastric contents in, 116
 gastro-intestinal atony and, 114, 116
 mental strain and, 114
 nephrolithiasis and, 114
 pain in, 115
 pathology of, 115
 prognosis of, 117
 symptoms of, 115
 treatment of, 118
 diet in, 118
 hygienic, 120
 medicinal, 121

- Hyperacidity, ulcer of stomach and, 114, 181
 urine in, 117
 vomiting in, 116
 Hyperæsthesia, diagnosis of, from gastric ulcer, 205
 gastrica, 138. *See* Gastric hyperæsthesia.
 of œsophagus, 86
 Hyperalgesia in appendicitis, 413
 Hyperchlorhydria, 111. *See* Hyperacidity.
 Hyperemesis lactentium, 303
 Hypernephroma, diagnosis of, from perigastric abscess, 543
 Hyperorexia, 133
 Hyperplastic chronic perihepatitis, 737
 tuberculosis of intestines, chronic, 437, 562
 Hyperptyalism, 68
 Hyperpyrexia in enterocolitis, 385
 Hypersecretion, chronic, diagnosis of, from hyperacidity, 117
 diagnosis of, from gastrectasis, 295
 of gastric juice, 122. *See* Gastro-succorrhœa.
 Hypertrophic stenosis of pylorus, congenital, 303
 Hypochlorhydria, 155
 definition of, 155
 diagnosis of, 156
 from gastric ulcer, 205
 symptoms of, 155
 treatment of, 156
 Hypocholia in jaundice, 697
 Hypotyalism, 69
 Hysteria, mucous colitis and, 493
 vomiting and, 146
 Hysterical vomiting, diagnosis of, from gastric ulcer, 205
- I**
- ICTERUS gravis, 690
 melas, 694
 neonatorum, 692
 etiology of, 692
 Ichthyosis buccalis, 65
 Idiopathic dilatation of colon, 363
 of stomach, 285
 Ileus, 443
 Ileum, carcinoma of, 480
 constipation in, 480
 diarrhœa in, 480
 hemorrhages in, 481
 pain in, 480
 Incarceration of intestines, 443
 Incontinence of pylorus, 165. *See* pylorus, incontinence of.
 Indicanuria in appendicitis, 418
 Indigestion, intestinal, 343. *See* Intestinal indigestion.
- Indigestion in splachnoptosis, 607
 Infantile hypertrophic stenosis of pylorus, 303
 Infection, digestion and, 32, 34
 Infective œsophagitis, 81
 Inflammation of biliary tract, cholelithiasis and, 827, 839
 of colon, 380. *See* Colitis.
 of mouth, 45. *See* Stomatitis.
 of œsophagus, 79. *See* (Esophagitis.
 of omentum, 541. *See* Epiploitis.
 of pericardium, chronic peritonitis and, 562
 of peritoneum, 513. *See* Peritonitis.
 of pleura, chronic peritonitis and, 562
 of stomach, 253. *See* Gastritis.
 Inflammatory diarrhœa in childhood, 384
 Influenza bacillus in appendicitis, 407
 intestinal colic and, 346
 Infrapapillary carcinoma, 480
 Insomnia in nervous dyspepsia, 172
 Interacinar pancreatitis, chronic, 654
 Intercostal neuralgia, diagnosis of, from gastralgia, 137
 Interlobular pancreatitis, chronic, 653
 Interstitial gastritis, chronic, 310
 purulent, 258
 hepatitis, chronic, 757
 pancreatitis, chronic, 650
 Intestinal colic, 345
 appendicular, 346
 constipation in, 347
 definition of, 345
 diagnosis of, 348
 from appendicitis, 424
 from neuralgia of intestines, 347
 diarrhœa in, 347
 etiology of, 345
 faecal accumulations and, 346
 functional disturbances and, 345
 influenza and, 345
 organic conditions and, 346
 pain in, 347
 peristalsis in, 347
 rheumatic, 346
 symptoms of, 347
 treatment of, 348
 diverticula, 472. *See* Intestines, diverticula of.
 fermentative dyspepsia, stools in, 344
 hemorrhage, 349
 amyloid degeneration and, 350
 anæmia and, 350
 congestive conditions of bowel and, 350
 diarrhœa in, 351
 disease of bloodvessels and, 350
 duodenal ulcer and, 350
 etiology of, 349
 fæces in, 350
 gastric ulcer and, 350

- Intestinal hemorrhage, hemorrhagic infarction of bowel and, 350
 hemorrhoids and, 350
 hernia and, 350
 inflammatory conditions of bowel and, 350
 interstitial nephritis and, 350
 intussusception and, 350
 malarial fever and, 349
 prognosis of, 351
 purpura and, 349
 scurvy and, 349
 septicopyæmia and, 349
 strangulation of bowel and, 350
 symptoms of, 350
 treatment of, 351
 unassociated with pathological conditions of bowel, 349
 valvular disease of heart and, 350
 yellow fever and, 349
- indigestion, 343
 diagnosis of, 344
 etiology of, 343
 pain in, 344
 stools in, 344
 symptoms of, 344
 treatment of, 344
- obstruction, 442. *See* Intestines, obstruction of.
- processes, automatic mechanism in, 332
- sand, 336
 false, 336
 in mucous colitis, 493
- torpor, constipation and, 354
- tract, pathogenic organisms in, 339
 physiology of, 327
- Intestines, absorption of carbohydrates by, 331
 of fats by, 332
 of food by, 331
 of proteids by, 332
- in acute peritonitis, 520
- adenocarcinoma of, 477
- adenoma of, 485
- adhesions of, carcinoma and, 478
- amyloid disease of, 439
- antiperistalsis of, 328
 pathological, 329
- arteriosclerosis of, 490
- atony of, gastric atony and, 168
- bacillus botulinus in, 339
 pyocyaneus in, 339
- bacteria in, clinical results of, 339
 defensive role of, 338
 distribution of, 336
 growth of, conditions influencing, 338
 influence of age on, 337
 significance of, 338
 treatment of, 340
- Intestines, bacteria in, virulence of, conditions influencing, 338
- bacteriology of, 336
- carcinoma of, 476
 adhesions and, 478
 age in, 476
 blood in, 483
 colloid, 477
 complications of, 483
 cylinder-celled, solid, 477
 diagnosis of, from intestinal diverticula, 475
 dilatation and, 478
 feces in, 483
 gastrocolic fistula in, 483
 intussusception in, 483
 medullary, 477
 metastasis and, 479
 obstruction and, 477
 pathology of, 477
 perforation and, 478
 into bladder in, 484
 pericolic abscess and, 543
 peritonitis and, 479, 483
 pus in, 483
 round-celled, solid, 477
 scirrhus, 477
 sequelæ of, 477
 sex in, 476
 simple, 477
 symptoms of, 479
 treatment of, 484
 tumor in, 482
 volvulus in, 483
- chemical processes in, 329
- cholera bacillus in, 339
- compression of, 443
- concretions of, obstruction and, 453
- constriction of, 443
- descent of, 612
 diagnosis of, 615
 sigmoidopexy in, 620
 symptoms of, 612
 treatment of, operative, 620
- dilatation of, carcinoma and, 477
- diseases of, 327
- distention of, in intestinal indigestion, 344
- diverticula of, 472
 acquired, 473
 congenital, 472
 diagnosis of, from carcinoma, 475
 false, 473
 symptoms of, 475
 treatment of, 475
 true, 472
- dysentery bacillus in, 339
- as excretory organs, 332
- gaseous distention of, 341
- hemangioma of, 486
- incarceration of, 443
- large, absorption in, 332
- lipoma of, 486

- Intestines, lymphangioma of, 486
 lymphosarcoma of, 484
 symptoms of, 485
 treatment of, 485
 motor functions of, 327
 movements of, mechanism of, 328
 myoma of, 486
 neuralgia of, diagnosis of, from intestinal colic, 347
 obstruction of, 442
 acute, 444
 diagnosis of, from appendicitis, 425
 bacillus coli in, 448
 carcinoma and, 477
 of pancreas and, 678
 cholelithiasis and, 841
 chronic, 443
 diagnosis of, from intestinal colic, 348
 collapse in, 449
 constipation in, 447
 definition of, 442
 diagnosis of, 450, 465
 from acute dilatation of stomach, 283
 pancreatitis, 644
 peritonitis, 532
 from ascites, 507
 from duodenal ulcer, 393
 from enterocolitis, 451
 from locomotor ataxia, 451
 from peritonitis, 451
 from phantom tumor, 451
 from pneumonia, 451
 from typhoid fever, 451
 situation of, 451
 urine in, 452
 diaphragmatic herniæ and, 455
 diarrhœa in, 447
 due to bands, 454
 fæcal masses, 452
 foreign bodies, 452, 453
 internal hernia, 455
 intussusception, 461
 diarrhœa in, 461
 distention in, 461
 hemorrhages in, 462
 pain in, 461
 prognosis of, 462
 symptoms of, 461
 tenesmus in, 462
 tumor in, 461
 vomiting in, 461
 knotting, 462. *See* Volvulus.
 paralysis of bowel, 469
 embolism and, 470
 meteorism and, 471
 pathological anatomy of, 471
 peritonitis and, 470
- Intestines, obstruction of, due to paralysis of bowel, postoperative, 469
 symptoms of, 471
 thrombosis and, 470
 treatment of, 471
 twisting, 462. *See* Volvulus.
 duodenojejunal hernia and, 455
 external hernia and, 455
 from adhesions, 456
 from compression, 458
 from congenital stenosis, 458
 from intussusception, 458
 age in, 460
 etiology of, 459
 location of, 460
 pathological anatomy of, 460
 from stricture, 457
 definition of, 457
 diagnosis of, 458
 pathological anatomy of, 458
 symptoms of, 458
 treatment of, 458
 functional paralysis and, 469
 gallstones and, 452
 symptoms of, 453
 herniæ into broad ligament of uterus and, 455
 into Douglas' pouch and, 455
 of foramen of Winslow and, 455
 by incarceration of bowel, 456
 intersigmoidal hernia and, 455
 intestinal concretions and, 453
 Meckel's diverticulum and, 454
 meteorism in, 446
 pain in, 445
 pathological anatomy of, 443
 pericæcal hernia and, 455
 prognosis of, 466
 symptoms of, 445
 treatment of, 467
 surgical, 468
 in tuberculosis of peritoneum, 573
 vomiting in, 478
 obturation of, 443
 occlusion of, 443
 paralysis of, in peritonitis, 525
 perforation of, carcinoma and, 478
 in gastric ulcer, 199
 peristalsis, increased, 329
 pathological, 329
 varieties of, 328
 proper, 327
 visible, 329
 in portal cirrhosis, 771
 rhythmic segmentation of, 327

Intestines, sarcoma of, 484
 symptoms of, 485
 treatment of, 485
 small, diseases of, acute peritonitis and, 516
 stenosis of, constipation and, 356
 obstruction due to, 458
 strangulation of, 443
 stricture of, 443
 obstruction due to, 457
 pericolic abscesses and, 544
 syphilis of, 439
 tonic contractions of, 329
 tubercle bacillus in, 339
 tuberculosis of, 434
 chronic hyperplastic, 437, 562
 stenosing, 437
 treatment of, 438
 tuberculous of peritoneum and, 569
 ulcerative, 435
 diagnosis of, 436
 from amyloid disease, 437
 feces in, 436
 symptoms of, 436
 terminal changes in, 436
 treatment of, 438
 tumors of, benign, 485
 symptoms of, 486
 treatment of, 487
 malignant, 476
 typhoid bacillus in, 339
 ulcers of, 388
 anæmia in, 398
 catarrhal, 395
 classification of, 388
 decubital, 396
 diagnosis of, 398
 diarrhœa in, 398
 duodenal, 389
 embolic, 395
 fever in, 398
 follicular, 396
 hemorrhage in, 398
 jejunal peptic, 394
 leukæmic, 397
 in mucous colitis, 493
 pericolic abscesses and, 543
 prognosis of, 399
 pus in, 398
 stercoral, 396
 symptoms of, 397
 thrombotic, 395
 treatment of, 399
 trophic, 397
 tuberculous of peritoneum and, 574
 uræmic, 394
 Intraperitoneal subphrenic abscess, 548
 Intussusception, 459. *See* Intestines,
 obstruction of, due to intussuscep-
 tion.

Intussusception, diagnosis of, from tuber-
 culosis of peritoneum, 575
 Inverting enzymes, 331
 Iodic parotitis, 70

J

JAUNDICE, 689
 in abscess of liver, 750, 751, 752
 in active congestion of liver, 727
 in acute catarrhal cholangitis, 809
 cholecystitis, 822, 823
 non-suppurative hepatitis, 744
 pancreatitis, 641, 642
 simple gastritis, 256
 yellow atrophy of liver, 721, 722
 in aneurism of hepatic artery, 702
 in biliary cirrhosis, 792
 black, 694
 blood in, 694
 bloodvessels in, 695
 boils in, 694
 bradycardia in, 695
 calculi of pancreas and, 660
 in carcinoma of biliary ducts, 854
 of gall-bladder, 853
 of liver, 797
 of pancreas and, 678, 679
 catarrhal, 690, 807. *See* Cholan-
 gitis, catarrhal, acute.
 diagnosis of, from acute yellow
 atrophy of liver, 723
 in cholelithiasis, 834
 in chronic peritonitis, 560, 566
 in circumpapillary carcinoma, 480
 in cirrhosis of stomach, 317
 coma in, 697
 congenital, 802
 convulsions in, 697
 in cysts of pancreas, 672
 delirium in, 697
 effects of, 693
 emotional, 693
 in endophlebitis of hepatic veins, 705
 erythrocytes in, 695
 etiology of, 689
 feces in, 698
 in fatty liver, 714
 gastro-intestinal tract in, 697
 grave, 690
 hæmatogenous, 690
 heart in, 695
 hemeralopia in, 697
 hepatogenous, 690
 hereditary, 693
 hypocholia in, 697
 infectious, 690
 diagnosis of, from acute catar-
 rhal cholangitis, 810
 liver in, 696
 menstrual, 693
 mucous membranes in, 693

Jaundice, nervous, 693
 system in, 697
 nutrition in, 698
 obstructive, 691
 oligocholelia in, 697
 in passive congestion of liver, 733
 pathogenesis of, 691
 phenol in urine in, 696
 polycythæmia in, 695
 in portal cirrhosis, 773, 774
 pruritus in, 694
 skin in, 693
 in suppurative cholangitis, 813
 pancreatitis, 648
 telangiectases of skin in, 694
 toxic, 69
 in toxic gastritis, 263
 in tuberculosis of peritoneum, 574
 types of, 689
 urine in, 695
 urobilinuria in, 696
 vitiligoidea in, 694
 xanthelasma in, 694
 xanthoma in, 694
 xanthopsia in, 697
 Jejunal peptic ulcer, 394
 Jejunum, carcinoma of, 480
 Juvenile vomiting, 147

K

KERATOSIS buccalis, 65
 Kidneys, abscess of, subphrenic abscess
 and, 547
 anatomy of, 600
 displacement of, 609. *See* Nephroptosis.
 gangrene of, in nephroptosis, 610
 in general chronic perihepatitis, 739
 movable, diagnosis of, from appendicitis, 427
 from gastric carcinoma, 244
 frequency of, 604
 gastrectasis and, 287
 in portal cirrhosis, 771
 pressure of, acute dilatation of stomach and, 282

L

LABIAL glands, inflammation of, 45
 Lacrymal glands, enlargement of, 72
 Lactescent ascites, 504
 Lactic acid in carcinoma of stomach, 237
 Laennec's cirrhosis of liver, 758
 Langerhans, islands of, in carcinoma of pancreas, 677
 Lardaceous disease of liver, ascites and, 500
 Larynx, carcinoma of, breath in, 42
 Lead colic, 349

Leprosy of mouth, 60
 Leukæmia, diagnosis of, from congestion of liver, 734
 Leukæmic infiltration of liver, 717
 ulcers of intestines, 397
 Leukocytosis in abscess of liver, 750, 751
 in acute cholecystitis, 822, 823
 pancreatitis, 638, 641
 in appendicitis, 416
 in hæmatemesis, 320
 in phlegmonous gastritis, 261
 in suppurative cholangitis, 814
 pancreatitis, 648
 Leukoplakia, 65
 etiology of, 65
 pathology of, 66
 relation of, to epithelioma, 66
 symptoms of, 66
 treatment of, 66
 Leukoplasia, 65
 Levuloseuria, alimentary, in hepatic cirrhosis, 688
 in portal cirrhosis, 779
 Lichen planus of mouth, 54
 Linguopapillitis, 63
 Lingua nigra, 62
 Lipase, 330
 Lipogenic function of liver, 686
 Lipoma of intestines, 486
 of liver, 801
 of peritoneum, 581
 Lips, color of, in Addison's disease, 44
 in anæmia, 44
 in aortic stenosis, 44
 in asphyxia, 44
 in chronic parenchymatous nephritis, 44
 in congenital heart disease, 44
 in emphysema, 44
 in hemorrhage, 44
 in intestinal toxæmia, 44
 in jaundice, 44
 in lead poisoning, 44
 in mediastinal tumor, 44
 in pneumonia, 44
 double, 43
 dryness of, 44
 eruptions of, 44
 fissure of, 44
 tremor of, 44, 45
 Lithæmic splanchnoptosis, 608
 Liver, abscess of, 745
 amœbæ in, 754
 appendicitis and, 411, 747
 biliary duct infection in, 748
 blood in, 754
 chills in, 750, 751, 752
 diagnosis of, 755
 from biliary intermittent fever, 756
 from carcinoma, 800
 from cholelithiasis, 756
 from malarial fever, 756

- Liver, abscess of, diarrhœa in, 754
 dysentery and, 746
 etiology of, 746
 facies in, 753
 fever in, 750, 751, 752
 hepatic artery infection in, 747
 vein infection in, 747
 jaundice in, 750, 751, 752
 liver in, 748, 753
 lymphatic infection in, 748
 microorganisms and, 746
 pain in, 750, 751, 752
 pathology of, 748
 prognosis of, 756
 rupture of, 754
 septicopyæmic, 751
 signs of, 753
 spleen in, 754
 subphrenic abscess and, 547
 suppurative cholecystitis and, 746
 in suppurative pancreatitis, 649
 sweating in, 752
 symptoms of, 750
 traumatic, 750
 traumatism and, 746
 treatment of, 756
 tropical, symptoms of, 751
 urine in, 753
 actinomycosis of, 795
 in acute catarrhal cholangitis, 809
 non-suppurative hepatitis, 744
 yellow atrophy of, 718
 alcohol and, 719
 ascites in, 723
 blood in, 723
 chemistry of, 721
 chloroform poisoning and, 719
 diagnosis of, 723
 from biliary cirrhosis, 723
 from catarrhal jaundice, 723
 from phosphorus poisoning, 723
 diarrhœa in, 722
 etiology of, 718
 faces in, 722
 hematemesis in, 722
 hemorrhages in, 721, 722
 histology of, 720
 hyperplasia in, 720
 jaundice in, 721, 722
 leucin in, 721
 microorganisms and, 719
 pathogenesis of, 721
 pathology of, 719
 phosphorus poisoning and, 719
 pregnancy and, 718
- Liver, acute yellow atrophy of, prog-
 nosis of, 724
 spleen in, 720, 722
 symptoms of, 722
 syphilis and, 719
 treatment of, 724
 tyrosin in, 721
 urine in, 721, 722
 adenoma of, 801
 amyloid, 714
 diagnosis of, 716
 from cirrhosis, 780
 from congestion, 734
 from enlargement of liver, 716
 from fatty liver, 714, 716
 etiology of, 714
 histology of, 715
 pathology of, 715
 prognosis of, 716
 spleen in, 715
 symptoms of, 715
 treatment of, 716
 anatomy of, 600
 angioma of, 801
 anomalies of form of, acquired, 699
 biliary colic in, 700
 diagnosis of, 700
 prognosis of, 700
 symptoms of, 700
 treatment of, 700
 congenital, 698
 of position of, acquired, 700
 diagnosis of, 701
 symptoms of, 701
 atrophy of, 710
 acute yellow, 718
 diagnosis of, 712
 etiology of, 711
 general, 711
 histology of, 711
 localized, 711
 metabolism and, 711
 pathology of, 711
 prognosis of, 712
 symptoms of, 711
 treatment of, 712
 in biliary cirrhosis, 791, 793
 carcinoma of, 795
 ascites in, 797
 of biliary ducts, 852
 diagnosis of, 799
 from abscess of liver, 800
 from biliary cirrhosis, 800
 from carcinoma of gall-bladder, 855
 of stomach, 244
 from hydatid disease of liver, 800
 from postal cirrhosis, 800
 from syphilis of liver, 800
 from tumors of omentum, 799

- Liver, carcinoma of, diagnosis of, from
tumors of stomach, 799
etiology of, 795
gall-bladder in, 798, 852
jaundice in, 797
pathology of, 796
prognosis of, 800
symptoms of, 797
treatment of, 800
- cardiac, 729
in chronic perihepatitis, 738, 739, 741
circulation of, 765
cirrhosis of, 757
anthracotic, 785
ascites and, 499
biliary, 790
diagnosis of, 794
from acute yellow atrophy of liver, 723
from carcinoma of liver, 800
from portal cirrhosis, 794
etiology of, 790
fever in, 793
forms of, 758
Hippocratic fingers in, 793
histology of, 791
jaundice in, 792
pathology of, 791
prognosis of, 795
spleen in, 792, 793
symptoms of, 792
treatment of, 795
types of, 793
- Budd's non-alcoholic, 760
in children, 785
chronic pancreatitis and, 652
diagnosis of, from chronic perihepatitis, 742
from congestion, 734
from fatty liver, 714
from portal thrombosis, 707
from peritoneal tuberculosis, 575
from tumors of peritoneum, 597
- dyspeptic, 760
forms of, 757
in hæmochromatosis, 783
intestinal hemorrhage and, 350
levulosuria in, 688
malarial, 707, 784
portal, 759
alcohol and, 760
anastomosis in, 763
ascites in, 776
bowels in, 773
in children, 785
coma in, 775
complications of, 779
connective tissue in, 769
convulsions in, 775
- Liver, cirrhosis of, portal, delirium in, 775
diagnosis of, 780
from amyloid liver, 780
from anæmia, 782
from biliary cirrhosis, 780, 794
from carcinoma of liver, 800
of stomach, 781
from chronic peritonitis, 781
from fatty liver, 780
from nephritis, 782
from passive congestion, 780
from portal thrombosis, 781
from splenic anæmia, 781
from syphilis, 780
from tuberculosis of peritoneum, 781
from tumors of peritoneum, 781
from ulcer of duodenum, 781
of stomach, 781
- etiology of, 759
fatty acids and, 760
fever in, 773, 775
hæmatemesis in, 773
hæmochromatosis and, 761
heart in, 778
hemorrhoids in, 773
histology of, 765
intestines in, 771
jaundice in, 774
kidneys in, 771
levulosuria in, 779
liver in, 761, 776
malarial fever and, 761
nephritis in, 780
neuritis in, 780
occult blood in, 774
œdema in, 778
operation in, 789
pain in, 773
pancreas in, 771
pathogenesis of, 771
pathology of, 761
peritoneum in, 771
phenoluria in, 779
prognosis of, 785
rectum in, 771
signs of, 775
spleen in, 770, 776
splenic anæmia and, 760
stomach in, 770
stupor in, 775
symptoms of, 773
syphilis and, 761
treatment of, 786

- Liver, cirrhosis of, portal, tuberculosis
 and, 761, 779
 types of, 783
 urine in, 779
 urobilinuria in, 779
 vomiting in, 773
 mesenteric thrombosis and, 488
 tuberculosis of peritoneum and, 568
 cloudy swelling of, 712
 congestion of, 725
 active, 726
 albuminuria in, 727
 "cold" and, 726
 diagnosis of, 727
 etiology of, 726
 jaundice in, 727
 pain in, 727
 pathology of, 727
 prognosis of, 728
 symptoms of, 727
 treatment of, 728
 urine in, 727
 passive, 729
 ascites in, 733
 circulation and, 730
 diagnosis of, 734
 from amyloid degenera-
 tion, 734
 from cirrhosis, 734, 780
 from leukæmia, 734
 from malignant dis-
 ease, 734
 in diseases of lungs, 730
 dyspnoea in, 734
 etiology of, 729
 heart in, 729, 731, 732
 histology of, 731
 jaundice in, 733
 œdema in, 733
 pain in, 733
 pathology of, 730
 peritonitis in, 733
 prognosis of, 734
 symptoms of, 732
 treatment of, 734
 urine in, 734
 corset, 699
 cysts of, 801
 hydatid, subphrenic pneumotho-
 rax and, 556
 diseases of, acute peritonitis and, 516
 displacement of, diagnosis of, from
 fatty liver, 714
 emphysematous, 712
 enlargement of, diagnosis of, from
 amyloid liver, 716
 from fatty liver, 714
 fatty, 712
 alcoholism and, 713
 anæmia and, 713
 cachexia and, 713
 diagnosis of, 714
 Liver, fatty, diagnosis of, from amyloid
 liver, 714, 716
 from cirrhosis, 714, 781
 from displacement of liver,
 714
 from enlargement of liver,
 714
 etiology of, 713
 histology of, 713
 infections and, 713
 jaundice in, 714
 metabolism and, 713
 pathology of, 713
 prognosis of, 714
 symptoms of, 714
 treatment of, 714
 vomiting in, 714
 fibroma of, 801
 floating, 612. *See* Hepatoptosis.
 functions of, 684
 detoxifying, 686
 disorders of, 683
 failure of, 685
 glycogenic, 687
 lipogenic, 686
 test of, 685
 urea-forming, 687
 gaseous, 712
 gumma of, diagnosis of, from carci-
 noma of stomach, 244
 in hæmochromatosis, 783
 hydatid disease of, diagnosis of, from
 carcinoma, 800
 infiltration of, calcareous, 717
 leukæmic, 717
 in jaundice, 696
 lardaceous disease of, ascites and, 500
 linguiform lobulation of, 699
 lipoma of, 801
 lymphatic glands of, diseases of, 710
 vessels of, diseases of, 710
 necrosis of, 717
 nutmeg, ascites and, 499
 parenchymatous degeneration of, 712
 perforation of, in gastric ulcer, 199
 pericarditic pseudocirrhosis of, 737
 pigmentation of, 716
 bilirubin in, 716
 biliverdin in, 716
 in hæmochromatosis, 716
 hæmofuscin in, 716
 hæmosiderin in, 716
 in malarial fever, 716
 in portal cirrhosis, 761
 retrogressive disorders of, 710
 rupture of, hæmoperitoneum and, 511
 sarcoma of, 801
 sugar-iced, 737
 in suppurative cholangitis, 813, 814
 syphilis of, 795
 ascites and, 500
 chronic peritonitis and, 563
 diagnosis of, from carcinoma, 800

- Liver in thrombosis of portal vein, 707
 tropical, 726
 tuberculosis of, 795
 tumors of, ascites and, 500
 benign, 801
 malignant, 795
- Locomotor ataxia, diagnosis of, from intestinal obstruction, 451
- Loreta's operation in congenital stenosis of pylorus, 310
- Ludwig's angina, 75
 symptoms of, 76
 treatment of, 76
- Lungs, diseases of, cholelithiasis and, 828
 in tuberculosis of peritoneum, 569
- Lupus erythematosus of mouth, 53
- Lycorexia, 133
- Lymphangioma of intestines, 486
 of lips, 45
- Lymphatic glands of liver, diseases of, 710
 vessels of liver, diseases of, 710
- Lymphatics in acute peritonitis, 520
 of appendix, 404
 in cirrhosis of stomach, 315
- M**
- McBURNey's point, 401
- Macrocheilia, 61
- Macroglossia, 61
- Malacia, 135
- Malarial cirrhosis of liver, 784
 fever, diagnosis of, from appendicitis, 426
 from suppurative cholangitis, 815
 diarrhoea and, 367
 intestinal colic and, 346
 hemorrhage and, 349
 pernicious, diagnosis of, from acute peritonitis, 534
 pigmentation of liver in, 716
 portal cirrhosis and, 761
- Malformations of œsophagus, 79
- Malnutrition in dilatation of colon, 365
- Meckel's diverticulum, 472
 intestinal obstruction and, 454
- Medullary carcinoma of stomach, 226
- Melena in thrombosis of portal vein, 707
- Melanoglossia, 62
- Membranous cholecystitis, 820
 colitis, 491
 gastritis, 265
- Meningitis, tuberculous, tuberculosis of peritoneum and, 575
- Menstrual jaundice, 693
- Mental strain, hyperacidity and, 114
- Mercurial stomatitis, 48
- Merycism, 139
 definition of, 140
 diagnosis of, 141
 etiology of, 140
- Merycism, prognosis of, 141
 symptoms of, 141
 treatment of, 141
- Mesenteric arteries, embolism of, 487
 abdomen in, 489
 atheroma and, 488
 diagnosis of, 490
 from acute peritonitis, 533
 diarrhoea in, 489
 endocarditis and, 487
 etiology of, 487
 hemorrhage in, 489
 pain in, 489
 pathological anatomy of, 488
 prognosis of, 490
 symptoms of, 489
 treatment of, 490
 vomiting in, 489
- thrombosis of, 487
 abdomen in, 489
 cirrhosis of liver and, 488
 diagnosis of, 490
 diarrhoea in, 489
 etiology of, 488
 hemorrhage in, 489
 pain in, 489
 pathological anatomy of, 488
 prognosis of, 490
 symptoms of, 489
 thrombosis of portal vein and, 488
 treatment of, 490
 vomiting in, 489
- glands, diseases of, acute peritonitis and, 516
 peritonitis, chronic, 561
- Mesentery, cysts of, 585
 diagnosis of, from cysts of pancreas, 673
 rupture of, hæmoperitoneum and, 511
 suppuration in, 542
 traction of, 37
- Meso-appendix, 402
- Mesosigmoiditis, 561
 symptoms of, 561
- Metabolism, atrophy of liver and, 711
 cholelithiasis and, 829
 fatty liver and, 713
- Metastases in carcinoma of intestines, 479
 of stomach, 227
- Meteorism, 340
- Microglossia, 61
- Mikulicz's disease, 72
- Milky ascites, 504
- Moeller's glossitis, 64
- Motor functions of intestines, 327
- Mouth, acne of, 54
 actinomycosis of, 60

- Mouth, angioneurotic œdema of, 53
 bacillus diphtherie in, 40
 gangrene pulpæ in, 40
 of glanders in, 41
 lepre in, 41
 maximus in, 40
 necrodenialis in, 40
 pseudodiphthericus in, 40
 pyocyaneus in, 40
 of rhinoscleroma in, 41
 tuberculosis in, 40
 bacteriology of, 39
 bacterium gingivitis in, 40
 carcinoma of, 52
 cladotrix buccalis in, 40
 cysts of, 52
 dermoid, 53
 dermatoses of, 53
 in diabetes, 55
 diseases of, 39
 dry, 69
 eczema of, 54
 erysipelas of, 54
 iodiococcus vaginatus in, 40
 leprosy of, 60
 leptotrix buccalis maxima in, 40
 innominata in, 40
 in leukæmia, 55
 lichen planus of, 54
 in locomotor ataxia, 56
 lupus erythematosus of, 53
 pemphigus of, 54
 in pernicious anæmia, 55
 pneumococcus in, 40
 in pregnancy, 56
 psoriasis of, 53
 scleroderma of, 54
 scleroma of, 61
 in scurvy, 55
 spirochaetes in, 41
 in sprue, 441
 staphylococcus pyogenes in, 40
 streptococcus brevis in, 40
 pyogenes in, 40
 syphilis of, 58, 59
 tinea circinata of, 54
 tuberculosis of, 57
 tumors of, 52
 in typhoid fever, 54
 in uræmia, 55
 urticaria of, 53
 in varicella, 56
 in variola, 56
- Movable kidney, diagnosis of, from appendicitis, 427
- Mucocele of appendix, 433
- Mucous colic, 491
 colitis, 490. *See* Colitis, mucous.
 blood supply and, 33
 diarrhœa, 491
 membrane in mucous colitis, 491
- Muscles in ascites, 501, 505
- Mycotic gastritis, 263
- Mycotic stomatitis, 49
- Myoma of appendix, 433
 of intestines, 486
 of œsophagus, 104
 of peritoneum, 580
- Myxoma of appendix, 433
 of peritoneum, 583
- ## N
- NAUSEA in achylia gastrica, 159
 in appendicitis, 412, 414
 nervosa, 149
 in nervous gastralgia, 136
 in phlegmonous gastritis, 260
 salivation in, 69
 in splachnoptosis, 607
 in toxic gastritis, 262
 in volvulus of omentum, 469
- Necrosis of appendix, 400
 of liver, 717
- Neerotic œsophagitis, 80
- Nephritic colic, diagnosis of, from gastralgia, 137
- Nephritis, chronic perihepatitis and, 738
 diagnosis of, from portal cirrhosis, 782
 interstitial, intestinal hemorrhage and, 350
 nephroptosis and, 610
 in portal cirrhosis, 780
- Nephrolithiasis, diagnosis of, from appendicitis, 427
 hyperacidity and, 114
- Nephroptosis, 609
 appendicitis and, 610
 congenital, 610
 diagnosis of, 614
 from distended gall-bladder, 615
 from fecal tumors, 615
 from hepatoptosis, 615
 from myoma of uterus, 615
 from ovarian cyst, 615
 tumor, 615
 dilatation of stomach and, 611
- Dietl's crises in, 610
- gangrene of kidney in, 610
- hæmaturia in, 610
- hydronephrosis in, 610
- nephritis in, 610
- nephropexy in, 620
- neurasthenia in, 610
- symptoms of, 610
- treatment of, operative, 619
- urination in, 610
- urine in, 610
- Nephropexy in nephroptosis, 620
- Nervous anorexia, 163
 dyspepsia, 170
 eructation, 143
 gastralgia, 135
 jaundice, 693

Nervous nausea, 149
 system and gastric secretion, 25
 vomiting, 144
 Neuralgia, intercostal, diagnosis of, from
 gastralgia, 137
 trigeminal, salivation in, 69
 Neurasthenia, atony of stomach and, 167
 chronic gastritis and, 272
 gastrica, 170. *See* Dyspepsia, ner-
 vous.
 in mucous colitis, 493
 in nephroptosis, 610
 in splanchnoptosis, 607
 vomiting and, 146
 Neuropathic hæmatemesis, diagnosis of,
 322
 Neuroretinitis in hæmatemesis, 320
 Neuroses, gastric, 105. *See* Gastric neu-
 roses.
 of œsophagus, 86
 motor, 87
 sensory, 86
 Neurotrophic disturbance, 28
 Noma, 48

O

OBESITY, diagnosis of, from ascites, 507
 Obstipation, 352
 Obturation of intestines, 443
 Occlusion of intestines, 443
 "Occult bleeding" in carcinoma of stom-
 ach, 231, 238
 in gastric ulcer, 192
 aloin test for, 194
 benzin test for, 194
 chemical tests for, 193
 spectroscopic test for,
 193
 Teichmann's test for,
 193
 value of examination
 for, 194
 Œdema, angioneurotic, of mouth, 53
 carcinoma of pancreas and, 678
 in enterocolitis, 385
 in general chronic perihepatitis, 741
 in passive congestion of liver, 733
 in portal cirrhosis, 778
 in thrombosis of portal vein, 707
 in tuberculosis of peritoneum, 572
 Œsophageal regurgitation, diagnosis of,
 from gastric regurgitation, 142
 Œsophagitis, acute, 79
 diagnosis of, 82
 etiology of, 79
 pathology of, 80
 sequelæ of, 82
 symptoms of, 81
 dysphagia in, 81
 pain in, 82
 thirst in, 82
 treatment of, 82

Œsophagitis catarrhal, 80
 chronic, 82
 pathology of, 83
 sequelæ of, 83
 symptoms of, 83
 treatment of, 84
 corrosive, 81
 exfoliative, 80
 follicular, 80
 infective, 81
 necrotic, 80
 phlegmonous, 80
 ulcerative, 80
 Œsophagus, actinomycosis of, 86
 auscultation of, 78
 carcinoma of, 97
 diagnosis of, 102
 dysphagia in, 101
 dyspnœa in, 101
 etiology of, 97
 extension of, 100
 pain in, 101
 pathology of, 98
 perforation in, 99
 prognosis of, 102
 ptosis in, 101
 site of, 99
 symptoms of, 100
 treatment of, 102
 congenital malformations of, 79
 description of, 77
 dilatation of, 90
 in cardiospasm, 150
 diagnosis of, 91
 from diverticula, 91
 from gastrectasis, 295
 from stenosis, 91
 dyspnœa in, 90
 etiology of, 90
 idiopathic, 85
 prognosis of, 91
 symptoms of, 90
 treatment of, 91
 diseases of, 77
 diverticula of, 91
 breath in, 42
 diagnosis of, 93
 from spasm, 88
 etiology of, 91
 pathology of, 91
 pressure, 91
 respiration in, 93
 symptoms of, 93
 treatment of, 94
 vomiting in, 93
 ectasia of, 90
 examination of, 78
 fibroma of, 103
 fibromyoma of, 104
 herpes of, 89
 inflammation of, 79. *See* Œsoph-
 agitis.
 myoma of, 104

- Oesophagus, neuroses of, 86
 motor, 87
 diagnosis of, 88
 etiology of, 87
 symptoms of, 88
 treatment of, 89
 sensory, diagnosis of, 87
 treatment of, 87
 non-malignant-growths of, 103
 palpation of, 78
 papilloma of, 104
 paralysis of, 89
 pemphigus of, 89
 percussion of, 78
 rupture of, 85
 sarcoma of, 103
 spasm of, 87
 diagnosis of, 88
 etiology of, 87
 symptoms of, 88
 treatment of, 89
 stenosis of, diagnosis of, 91
 from spasm, 88, 96
 non-malignant, 94
 congenital, 95
 diagnosis of, 96
 from carcinoma, 96
 from foreign bodies, 96
 x-rays in, 96
 etiology of, 95
 fibrous, 95
 prognosis of, 96
 sequelæ of, 95
 symptoms of, 95
 treatment of, 96
 stricture of, non-malignant, 94
 syphilis of, 86
 tuberculosis of, 86
 ulcers of, 84
 decubitus, 84
 peptic, 84
 pressure, 84
 symptoms of, 84
 treatment of, 85
 uræmic, 85
 varices of, diagnosis of, from hæma-temesis, 322
 Oidium albicans, 46, 50
 Oligocholia in jaundice, 697
 Omentum, abscess of, 542
 cysts of, 587
 in general chronic perihepatitis, 739, 741
 inflammation of, 541. *See* Epiplo-itis.
 tumors of, diagnosis of, from carci-noma of liver, 799
 of stomach, 243
 volvulus of, 469
 Oöphoritis, diagnosis of, from appendi-citis, 428
 Optic neuritis in hæmatemesis, 320
 Oral sepsis, 41
 Organic disease, function and, 23, 28
 Ovarian cysts, acute peritonitis and, 516
 chronic peritonitis and, 560
 diagnosis of, from ascites, 507
 Ovary, cystadenoma of, diagnosis of, from hydatid cysts of peritoneum, 584
 cysts of, diagnosis of, from cysts of pancreas, 673
 from nephroptosis, 615
 neuralgia of, diagnosis of, from ap-pendicitis, 428
 tumors of, diagnosis of, from neph-roptosis, 615
 Ozæna, breath in, 42
- P**
- PAIN in abscess of liver, 750, 751, 752
 in acute cholecystitis, 821
 enteritis, 374
 non-suppurative hepatitis, 744
 pancreatitis, 640
 perihepatitis, 736
 peritonitis, 522, 524
 in appendicitis, 412
 in arteriosclerosis of intestines, 490
 in ascites, 506
 in calculi of pancreas, 660
 in carcinoma of biliary ducts, 855
 of colon, 481
 of gall-bladder, 853
 of ileum, 480
 of jejunum, 480
 of liver, 797
 of pancreas, 679
 in cholelithiasis, 833, 834
 in cholera nostras, 375
 in chronic dilatation of stomach, 289
 interstitial pancreatitis, 655
 peritonitis, 560
 in cirrhosis of stomach, 316
 in colitis, 380
 in congestion of liver, 727
 in constipation, 357
 in cysts of pancreas, 672
 in diarrhœa, 369
 in duodenal ulcer, 390
 in gastric ulcer, 188
 cause of, 189
 effect of food on, 189
 time of, 189
 in hæmatemesis, 320
 in intestinal colic, 347
 diverticula, 475
 indigestion, 344
 obstruction, 445
 due to paralysis of bowel, 471
 ulcers, 398
 in intussusception, 461
 in lead colic, 349

- Pain in mesenteric embolism, 489
 in thrombosis, 489
 in mucous colitis, 492
 in nephroptosis, 610
 in passive congestion of liver, 733
 in portal cirrhosis, 773
 in splanchnoptosis, 607
 in subphrenic abscess, 553
 pneumothorax, 558
 in suppurative cholangitis, 813
 pancreatitis, 648
 in tuberculosis of peritoneum, 572
 in volvulus, 464
 of omentum, 469
- Palate, soft, spasm of, 63
- Palpitation of heart in nervous dyspepsia, 172
- Pancreas, abscess of, 648. *See* Pancreatitis, suppurative.
 calculi of pancreas and, 661
 subphrenic abscess and, 547
 in acute dilatation of stomach, 282
 adenoma of, 677
 anatomy of, 600
 atrophy of, chronic interstitial pancreatitis and, 653
 calculi of, 657
 abscess of pancreas in, 661
 azotorrhœa in, 661
 bacillus coli in, 658
 carcinoma of pancreas in, 661
 cholelithiasis and, 657
 colic in, 660
 complications of, 661
 constituents of, 658
 cysts of pancreas and, 667
 diabetes mellitus in, 660
 diagnosis of, 661
 duct of Wirsung in, 659
 etiology of, 657
 glycosuria, alimentary in, 661
 jaundice in, 660
 pain in, 660
 pathology of, 658
 steatorrhœa in, 661
 symptoms of, 659
 treatment of, 662
 carcinoma of, 676
 ascites and, 678, 680
 bowels in, 679
 calculi of pancreas and, 661
 carcinoma of duodenum and, 678, 681
 of stomach and, 678, 681
 chronic pancreatitis and, 651
 complications of, 677
 treatment of, 682
 diabetes mellitus and, 677
 diagnosis of, 681
 from carcinoma of gall-bladder, 856
 from cholelithiasis, 681
- Pancreas, carcinoma of, diagnosis of,
 from circumpapillary carcinoma, 480
 from suppurative pancreatitis, 650
 fatty stools in, 679
 gall-bladder in, 680
 glycosuria in, 680
 hydronephrosis and, 678
 incidence of, 676
 intestinal obstruction and, 678
 islands of Langerhans in, 677
 jaundice and, 678, 679
 liver in, 680
 œdema and, 678
 pain in, 679
 pathology of, 676
 secondary, 676
 symptoms of, 678
 treatment of, 682
 tumor in, 679
 in cholelithiasis, 839
 cysts of, 663
 ascites in, 672
 classification of, 663
 contents of, 669
 diabetes mellitus in, 672
 diagnosis of, 673
 from aortic aneurism, 674
 from ascites, 507
 from cysts of mesentery, 673
 from distended gall-bladder, 673
 from echinococcus cysts, 673
 from hydronephrosis, 674
 from ovarian cysts, 673
 from suppurative pancreatitis, 650
 etiology of, 666
 ferments in, 670
 hemorrhagic, 669
 jaundice in, 672
 pain in, 672
 pancreatitis and, 643, 669
 pathology of, 666
 positions of, 664, 665
 proliferation, 668
 pseudocysts, 663
 relation of, to adjacent organs, 671
 retention, 667
 steatorrhœa in, 672
 symptoms of, 670
 traumatic, 668
 treatment of, 674
 true, 663
 tumors in, 670
 disappearance of, 671
 vomiting in, 672
 descent of, 613
 diseases of, 621

- Pancreas**, diseases of, alimentary glycosuria and, 628
 azotorrhœa in, 624
 diabetes mellitus and, 626
 due to impairment of metabolic function, 626
 of pancreatic secretion, 621
 feces in, 622, 624
 glycosuria and, 626
 pathology of, 621
 steatorrhœa in, 621
 symptoms of, 621
 in hæmochromatosis, 783
 hemorrhage into, 634
 perforation of, in gastric ulcer, 199
 in portal cirrhosis, 771
 sarcoma of, 677
 syphilis of, 656
 tuberculosis of, 656
- Pancreatic apoplexy**, 634
 cysts, acute pancreatitis and, 643
 juice, ferments of, 330
 secretion of, 329
- Pancreatitis**, 35
 acute hemorrhagic, 633
 abdomen in, 641, 642
 bacillus coli in, 636
 classification of, 635
 complications of, 643
 constipation in, 641
 course of, 639
 diabetes in, 643
 diagnosis of, 644
 from acute peritonitis, 533
 from appendicitis, 426
 from biliary colic, 644
 from duodenal ulcer, 393
 from intestinal obstruction, 644
 from perforation of gall-bladder, 644
 from ruptured aneurism, 644
 from suppuration of lesser peritoneum, 644
 from suppurative pancreatitis, 650
 duodenitis and, 635
 etiology of, 635
 feces in, 642
 fat necrosis and, 631
 fever in, 641, 642
 foudroyant, 640
 gastritis and, 635
 gastroduodenitis and, 636
 glycosuria in, 641, 642
 jaundice in, 641, 642
 pain in, 640
 pancreatic cysts and, 643
- Pancreatitis**, acute hemorrhagic, perforation of duodenum in, 643
 of stomach in, 643
 of transverse colon in, 643
 peripancreatic cysts and, 643
 pregnancy and, 636
 sequelæ of, 643
 shock in, 640
 stages of, 640
 symptoms of, 639
 thrombosis in, 643
 treatment of, 644
 tumor in, 642
 vomiting in, 640
- cholelithiasis** and, 839
- chronic interstitial**, 650
 alcoholism and, 652
 arteriosclerosis and, 652
 atrophy of pancreas and, 653
 carcinoma of pancreas and, 651
 cholelithiasis and, 651
 cirrhosis of liver and, 652
 diabetes and, 652, 655, 656
 diagnosis of, 655
 etiology of, 651
 glycosuria in, 655
 interacinar, 654
 interlobular, 653
 occlusion of duct of Wirsung and, 651
 pain in, 655
 pathology of, 653
 symptoms of, 655
 syphilis and, 652
 vomiting in, 655
- experimental**, 636
- gangrenous**, 638, 641
 fat necrosis and, 630
- suppurative**, 646
 abdomen in, 648
 abscess of liver in, 649
 bacteriology of, 647
 chills in, 648
 collapse in, 648
 definition of, 646
 diagnosis of, 649
 from acute hemorrhagic pancreatitis, 650
 emaciation in, 649
 etiology of, 646
 fever in, 648
 glycosuria in, 649
 jaundice in, 648
 leukocytosis in, 648
 pain in, 648
 pathology of, 647
 perforation of duodenum in, 649
 of stomach in, 649

- Pancreatitis, suppurative, peritonitis in,
649
 sequele of, 649
 symptoms of, 647
 thrombosis of splenic vein in, 649
 treatment of, 650
 tumor in, 648
 urine in, 649
 vomiting in, 648
traumatic, 636
 cholelithiasis and, 637, 643
 leukocytosis in, 638, 641
 pathology of, 637
- Panphlebitis, 709
- Papillitis lingualis, 63
- Papilloma of œsophagus, 104
- Paracentesis abdominis, 510
- Paracolon bacillus in intestines, 339
- Paræsthesia of œsophagus, 86
- Paragusia, 43
- Paralysis of bowel, intestinal obstruction
 due to, 469
 of œsophagus, 89
 of soft palate, 63
 of tongue, 62
- Paratyphoid bacillus in intestines, 339
- Parenchymatous degeneration of liver,
712, 743
- Parorexia, 135
- Parotid gland, cysts of, 52
- Parotitis, 70
 gastric ulcer and, 202
 iodic, 70
 in subphrenic pneumothorax, 558
- Pathogenic organisms in intestinal tract,
339
- Pemphigus of mouth, 54
 of œsophagus, 89
- Peptic ulcer, 175
 jejunal, 394
 of œsophagus, 84
- Peptonuria in carcinoma of stomach, 232
- Perforation in duodenal ulcer, 210, 390,
391
 in gastric ulcer, 197
 course of, 199
 into abdominal wall, 199
 into diaphragm, 199
 into gall-bladder, 199
 into intestines, 199
 into liver, 199
 into pancreas, 199
 into pericardium, 199
 into pleura, 199
 into portal vein, 199
 into spleen, 198
 pain in, 200
 signs of, 199
 symptoms of, 199
 treatment of, surgical, 219
- Peri-appendicular abscess, 410, 421
 rupture of, into bladder, 411
 into cæcum, 411
- Peri-appendicular abscess, rupture of,
 into gall-bladder, 411
 into peritoneal cavity, 411
 into pleural cavity, 411
 into ureter, 411
 into uterus, 411
 through abdominal wall, 411
- Pericarditic pseudocirrhosis, 499, 737
- Pericarditis in chronic perihepatitis, 741
 peritonitis and, 562
- Pericardium in chronic perihepatitis, 739
 perforation of, in gastric ulcer, 199
- Pericholecystic adhesions, chronic, 839
- Pericolic abscesses, 543
 diagnosis of, 544
 etiology of, 543
 subphrenic abscess and, 547
 symptoms of, 544
 treatment of, 544
- Pericolitis, 399
 acute, 540
 etiology of, 540
 symptoms of, 541
 treatment of, 541
 chronic, 561
 diagnosis of, 400
 from carcinoma, 400
 etiology of, 399
 pathology of, 399
 sinistra, 400
 symptoms of, 400
 treatment of, 400
- Perigastric abscess, 542
 definition of, 542
 diagnosis of, 543
 etiology of, 542
 signs of, 543
 symptoms of, 543
- Perigastritis, diagnosis of, from carcinoma
 of stomach, 243
 gastric ulcer and, 196
 pyloric, 197
 symptoms of, 197
 phlegmonous gastritis and, 259
- Parihepatitis, 735
 acute, 735
 ascites in, 736
 diagnosis of, 736
 etiology of, 735
 fever in, 736
 hiccough in, 736
 pain in, 736
 pathology of, 735
 prognosis of, 736
 symptoms of, 736
 treatment of, 737
 vomiting in, 736
 chronic, general, 737
 ascites in, 741
 arteriosclerosis and, 738, 741
 chronic interstitial nephri-
 tis and, 738
 diagnosis of, 741

- Perihepatitis, chronic, general, diagnosis of, from cirrhosis of liver, 742
 from malignant disease of liver, 742
 from tuberculous peritonitis, 742
 etiology of, 738
 histology of, 740
 infections and, 738
 kidneys in, 739
 liver in, 738, 739, 741
 oedema in, 741
 onset of, 741
 omentum in, 739, 741
 pain in, 741
 pathology of, 738
 pericarditis in, 741
 pericardium in, 739
 peritoneum in, 740
 pleura in, 739, 740
 pleuritis in, 741
 prognosis of, 742
 spleen in, 739, 741
 symptoms of, 740
 syphilis and, 738
 treatment of, 742
 tubercle bacillus and, 738
 hyperplastic, 737
 local, 737
 deforming, 737
 diagnosis of, from portal cirrhosis, 781
 hyaline, 737
 Periodical vomiting, 146
 in children, 147
 Peripancreatic cysts, acute pancreatitis and, 643
 Perisplenitis in gastric ulcer, 199
 Peristalsis in cirrhosis of stomach, 317
 in congenital stenosis of pylorus, 307
 gastric, 106
 increased, 329
 in intestinal colic, 347
 pathological, 328
 visible, 329
 Peristaltic unrest, gastric, 154
 definition of, 154
 diagnosis of, 155
 etiology of, 154
 symptoms of, 154
 treatment of, 155
 Peritoneal abscesses, residual, 544
 adhesions, intestinal obstruction due to, 456
 Peritoneum, absorption from, 495
 in acute peritonitis, 519
 adhesions of, 36
 angioma of, 582
 in chronic perihepatitis, 740
 comparison of, with other serous membranes, 497
 cysts of, allantoic, 590
 Peritoneum, cysts of, conditions simulating, 590
 cysticercus cellulosae, 585
 dermoid, 588
 hydatid, 583
 clinical aspects of, 584
 diagnosis of, 584
 from ovarian cystadenomas, 584
 from uterine fibromyoma, 584
 etiology of, 583
 morbid anatomy of, 583
 treatment of, 585
 mesenteric, 585
 diagnosis of, 586
 pathogeny of, 585
 pathological anatomy of, 586
 physical signs of, 586
 symptoms of, 586
 treatment of, 586
 omental, 587
 diagnosis of, 587
 symptoms of, 587
 retroperitoneal, 589
 sanguineous, 587
 clinical features of, 588
 diagnosis of, 588
 etiology of, 587
 pathology of, 588
 symptoms of, 588
 treatment of, 588
 urachal, 589
 treatment of, 590
 diseases of, 495
 endothelioma of, 591
 fibroma of, 579
 fibromyoma of, 580
 hemorrhage into, diagnosis of, from acute peritonitis, 532
 immunity of, 492, 536
 inflammation of, 513. *See* Peritonitis.
 lipoma of, 581
 diagnosis of, 582
 etiology of, 581
 physical signs of, 581
 prognosis of, 582
 symptoms of, 582
 treatment of, 582
 lymphatics of, 496
 myoma of, 580
 myxoma of, 583
 overtension of, 36
 in portal cirrhosis, 771
 pseudomyxoma of, 590
 retroperitoneal hematoma of, 588
 sarcoma of, 591
 traction on, 36
 tuberculosis of, 567
 abdomen in, 572, 573
 adhesive, 571

- Peritoneum, tuberculosis of, age in, 568
 albuminuria in, 572
 alcoholism and, 568
 ascitic, 570
 bacteriology of, 569
 blood in, 574
 chronic fibroid, 571
 cirrhosis of liver and, 568
 complications of, 574
 diagnosis of, 575
 from appendicitis, 427
 from carcinoma of stomach, 243
 from chronic perihepatitis, 575, 742
 from cirrhosis of liver, 575
 of stomach, 318
 from intussusception, 575
 from portal cirrhosis, 781
 from tumor of peritoneum, 525, 597
 disposing factors in, 568
 effusion in, 572
 encysted, 571
 etiology of, 568
 face in, 573
 faces in, 574
 fever in, 574
 fibrous, 571
 intestinal obstruction in, 573
 tuberculosis in, 569
 jaundice in, 574
 loculated, 571
 lungs in, 569
 morbid anatomy of, 570
 obliterative, 571
 oedema in, 572
 onset of, 572
 pain in, 572
 pregnancy and, 568
 prognosis of, 575
 pseudotuberculosis, 571
 pulse in, 574
 sex in, 568
 symptoms of, 572
 treatment of, 576
 hygienic, 577
 medicinal, 576
 surgical, 577
 vaccines in, 577
 tuberculosis of Fallopian tubes and, 569
 tumor in, 573
 tumors of, 579
 diagnosis of, from carcinoma of stomach, 243
 from portal cirrhosis, 781
 malignant, 591
 primary, 591
 ascites in, 593
 clinical picture of, 593
 diagnosis of, 593
 etiology of, 592
- Peritoneum, tumors of, malignant, primary, morbid anatomy of, 591
 prognosis of, 594
 treatment of, 594
 secondary, 594
 abdomen in, 596
 diagnosis of, 597
 duration of, 597
 pathology of, 594
 physical signs of, 596
 prognosis of, 598
 symptoms of, 596
 treatment of, 598
 non-malignant, 579
 cystic, 583
 solid, 579
- Peritonitis, acute cholecystitis and, 823
 circumscribed, 539
 non-suppurative, 539
 symptoms of, 540
 treatment of, 540
 diagnosis of, from appendicitis, 426, 427
 diffuse, 513
 abdomen in, 522
 abscess and, 516
 after-results of, 522
 age in, 517
 bacteriology of, 517
 blood in, 527
 cultures in, 528
 clinical picture of, 523
 constipation in, 526
 course of, 522
 cytology in, 521
 definition of, 513
 diagnosis of, 531
 age in, 534
 as to cause, 534
 from abdominal aneurism, 533
 from acute enterocolitis, 532
 pancreatitis, 533
 from Addison's disease, 534
 from colic, 532
 from enteralgia, 532
 from hemorrhage into peritoneum, 532
 from intestinal obstruction, 532
 from mesenteric embolism, 533
 from pernicious malaria, 534
 from pleurisy, 533
 from pneumonia, 533
 from rupture of ectopic gestation, 533
 from tympanites, 532

- Peritonitis, acute diffuse, diagnosis of, from uræmia, 534
sex in, 534
diseases of appendix and, 516
of bladder and, 516
of colon and, 516
of gall-bladder and, 516
of liver and, 516
of mesenteric glands and, 516
of small intestines and, 516
of spleen and, 516
of stomach and, 516
duration of, 528
etiology of, 515
exudate in, 519
facies in, 523
fever in, 527
hiccough in, 526
intestines in, 520
lymphatics in, 520
morbid anatomy of, 519
onset of, 522, 523
pain in, 522, 524
pathogeny of, 514
peritoneum in, 519
position in, 523
primary, 515
prognosis of, 535
pulse in, 527
pyosalpinx and, 516
respiration in, 526
rigidity in, 524
rigors in, 526
secondary, 515
sex in, 517
symptoms of, 522
tenderness in, 524
thirst in, 522
treatment of, 535
 laparotomy in, 537
 medical, 538
 prophylactic, 535
 serum, 539
 surgical, 536
 transfusion in, 538
in tuberculosis of peritoneum, 574
tympanites in, 525
urine in, 528
uterus and, 516
vomiting in, 522, 526
appendicitis and, 410, 411, 418
ascites and, 500
carcinoma of intestines and, 479, 483
chronic, ascites and, 498
 diagnosis of, from portal cirrhosis, 781
 diffuse, 562
 localized, 560
 appendicitis and, 560
Peritonitis, chronic, localized, cholecystitis and, 560
 constipation in, 561
 duodenal ulcer and, 560
 hydatid cysts and, 560
 etiology of, 560
 gastric ulcer and, 560
 gonococcal infections and, 560
 jaundice in, 560
 ovarian cysts and, 560
 pain in, 560
 puerperal infections and, 560
 symptoms of, 560
mesenteric, 561
of mesocolon of sigmoid flexure, 561
simple, 559
 abdomen in, 566
 adhesions and, 565
 alcoholism and, 563
 arteriosclerosis and, 563
 ascites and, 564, 566
 clinical picture of, 566
 diagnosis of, 567
 from tuberculosis of peritoneum, 575
 from tumors of peritoneum, 597
 etiology of, 562
 inflammation of pericardium and, 562
 of pleura and, 562
 jaundice in, 566
 morbid anatomy of, 564
 onset of, 566
 pathogeny of, 564
 prognosis of, 567
 respiration in, 567
 symptoms of, 566
 syphilis of liver and, 563
 traumatism and, 564
 treatment of, 567
deformans, 565
in duodenal ulcer, 392
gastric ulcer and, 197, 198
gonococcal, 530
intussusception and, 461
malignant, 594
in passive congestion of liver, 733
pneumococcal, 529
progressive fibropurulent, 520
puerperal, 531
in suppurative pancreatitis, 649
tuberculous, 567. *See* Peritoncum, tuberculosis of.
volvulus and, 463
Petechiæ in toxic gastritis, 263
Pfehl's sign in subphrenic abscess, 555
Phantom tumor, 342
 diagnosis of, from intestinal obstruction, 451

- Phlebitis, suppurative, of hepatic vein, 704
- Phlegmonous cholecystitis, 820
gastritis, 258
oesophagitis, 80
stomatitis, 47
- Phenoluria in portal cirrhosis, 779
- Phosphorus poisoning, acute yellow atrophy of liver and, 719
diagnosis of, from acute hepatitis, 745
from acute yellow atrophy of liver, 723
- Pica, 135
- Pigmentation of liver, 716
- Plastic linitis, 310
- Pleura in chronic perihepatitis, 739, 740
inflammation of, chronic peritonitis and, 562
perforation of, in gastric ulcer, 199
- Pleural effusion, diagnosis of, from subphrenic abscess, 554
pneumothorax, 559
- Pleurisy, diagnosis of, from acute peritonitis, 533
tuberculosis of peritoneum and, 574
- Pleuritis in chronic perihepatitis, 741
- Pneumatosis, 153
definition of, 153
diagnosis of, 154
from angina pectoris, 154
etiology of, 153
symptoms of, 154
treatment of, 154
- Pneumococcal peritonitis, 529
- Pneumococcus in mouth, 40
- Pneumonia, diagnosis of, from acute peritonitis, 533
from appendicitis, 426
from intestinal obstruction, 451
diarrhoea and, 367
- Pneumothorax, subphrenic, 555. *See* Subphrenic pneumothorax.
- Polycholia in jaundice, 692
- Polychromia in jaundice, 692
- Polycythæmia in jaundice, 695
- Polyorrhmenitis, 737
- Polyphagia, diagnosis of, from acoria, 163
from bulimia, 134
- Polyps of appendix, 433
- Polysymptomatic gastric neuroses, 170
- Portal cirrhosis, 759. *See* Liver, cirrhosis of, portal.
vein, chronic endophlebitis of, 710
dilatation of, 705
diseases of, 705
embolism of, 708
perforation of, in gastric ulcer, 199
pylephlebitis of, 708
chills in, 709
diagnosis of, 709
etiology of, 708
- Portal vein, pylephlebitis of, hamatemesis in, 709
pathology of, 709
prognosis of, 709
spleen in, 709
sweats in, 709
symptoms of, 709
treatment of, 710
- thrombosis of, 705
ascites and, 500, 707
diagnosis of, 707
from cirrhosis of liver, 707, 781
from tumors of peritoneum, 598
etiology of, 706
hamatemesis in, 707
in intrahepatic radicles of, 706
in main trunk of, 706
melæna in, 707
mesenteric thrombosis and, 488
œdema in, 707
pathology of, 706
in peripheral radicles of, 706
prognosis of, 708
spleen in, 707
symptoms of, 707
treatment of, 708
- Precardiac perigastritis, gastric ulcer and, 197
- Pregnancy, acute pancreatitis and, 636
yellow atrophy of liver and, 718
cholelithiasis and, 827
reflex vomiting in, 147
salivation in, 69
splachnoptosis and, 605
tuberculosis of peritoneum and, 568
with hyperemesis, diagnosis of, from gastric ulcer, 205
- Proctitis, gangrenous epidemic, 442
- Proliferation cysts of pancreas, 668
- Prosecretin, 330
- Proteids, absorption of, by intestines, 332
- Protozoa, diarrhoea and, 368
- Pruritus in jaundice, 694
- Pseudocirrhosis, pericarditic, 499, 737
- Pseudocysts of pancreas, 663
- Pseudodiphtheria bacillus in mouth, 40
- Pseudomyxoma of peritoneum, 590
- Pseudoperforation of gastric ulcer, 200
- Psilosis, 440. *See* Sprue.
- Psoriasis linguae, 65
of mouth, 53
- Psychic vomiting, 147
- Psychoneuroses, digestion and, 37
- Ptois in carcinoma of œsophagus, 101
- Ptyalism, 68
- Puerperal infections, cholelithiasis and, 828
chronic peritonitis and, 560
peritonitis, 531

- Pulmonary embolism in subphrenic abscess, 553
- Pulse in acute peritonitis, 527
in appendicitis, 416
in lead colic, 349
in phlegmonous gastritis, 260
in tuberculosis of peritoneum, 574
- Purpura, intestinal hemorrhage and, 349
- Pus in intestinal ulcers, 398
- Putrid sore mouth, 47
- Pyæmia in appendicitis, 419
portal, 708
- Pyelitis, acute, diagnosis of, from appendicitis, 427
- Pylephlebitis, 708
adhesiva, 705
in gastric ulcer, 199
- Pylethrombosis, 705
- Pylorotomy in congenital stenosis of pylorus, 310
- Pyloric perigastritis, gastric ulcer and, 197
stenosis, gastric ulcer and, 202
- Pyloroplasty in congenital stenosis of pylorus, 310
in gastrectasis, 303
in gastric ulcer, 218
- Pylorospasm, 152, 297
chronic gastrosuccorrhœa in, 153
congenital, 303
definition of, 152
diagnosis of, 153
etiology of, 152
symptoms of, 152
treatment of, 153
vomiting in, 153
- Pylorus, carcinoma of, diagnosis of, from duodenal ulcer, 393
from suprapapillary carcinoma, 479
in cirrhosis of stomach, 314
fibroid disease of, 310
incontinence of, 165
obstruction of, in circumpapillary carcinoma, 480
resection of, in gastrectasis, 303
spasm of, acute dilatation of stomach and, 282
diagnosis of, from carcinoma, 245
stenosis of, 296
cicatricial, 297
congenital hypertrophic, 303
abdomen in, 307
anorexia in, 306
bowels in, 307
diagnosis of, 308
dilatation in, 307
divulsion in, 310
gastro-enterostomy in, 310
history of, 303
morbid anatomy of, 304
- Pylorus, stenosis of, congenital hypertrophic, pain in, 307
pathogenesis of, 304
peristalsis in, 307
prognosis of, 308
pylorotomy in, 310
pyloroplasty in, 310
pylorospasm and, 304
signs of, 307
symptoms of, 306
treatment of, 309
true, 304
tumor in, 307
urine in, 307
vomiting in, 306
diagnosis of, 296
from external pressure, 297
foreign bodies and, 297
from neoplasm, 298
hypertrophic, 297, 310
diagnosis of, from carcinoma of stomach, 242, 245
perigastric adhesions with, 297
physical signs of, 296
varieties of, 297
ulcer of, diagnosis of, from carcinoma of stomach, 242, 244
- Pyopneumothorax in gastric ulcer, 199
- Pyorrhœa alveolaris, 56
diagnosis of, 57
etiology of, 56
symptoms of, 57
treatment of, 57
- Pyosalpinx, acute, peritonitis and, 516
- Pyrosis in achylia gastrica, 159
in atony of stomach, 167
in gastric ulcer, 188

R

- Rabies, virus of, 41
- Ranula pancreatica, 667
- Rash, wandering, of tongue, 63
- Rectum, prolapse of, in enterocolitis, 385
- Reflex vomiting, 147
- Regurgitation, gastric, 141
- Renal colic, diagnosis of, from intestinal colic, 348
disease, diarrhœa and, 367
dropsy, ascites and, 500
tumors, diagnosis of, from hepatoptosis, 615
- Residual peritoneal abscesses, 544
- Respiration in acute peritonitis, 526
in chronic peritonitis, 567
in diverticula of œsophagus, 93
- Retention cysts of pancreas, 667
- Retropertitoneal abscess, 544
definition of, 544
diagnosis of, 545

Retroperitoneal abscess, etiology of, 544
 symptoms of, 545
 treatment of, 545
 cysts, 589
 hæmatoma, 588
 Rheumatic colic, 346
 Rhinoscleroma, bacillus of, in mouth, 41
 Riga's disease, 66, 67
 Rigors in acute peritonitis, 526
 Round ulcer, 175
 Rumination, 139. *See* Merycism.
 Rupture of abscess of liver, 754
 of œsophagus, 85
 Rhythmic segmentation of intestines, 327

S

Saccharomyces albicans buccalis, 50
 Saliva, composition of, 67
 secretion of, 68
 Salivary calculi, 72, 73
 etiology of, 73
 symptoms of, 73
 treatment of, 74
 concretions, 72
 glands, atrophy of, 72
 diseases of, 67
 enlargement of, 72
 inflammation of, 70
 stone in, 73
 syphilis of, 72
 tuberculosis of, 71
 tumors of, 74
 Salivation, 68
 in children, 69
 Salpingitis, diagnosis of, from appendicitis, 428
 Sanguineous cysts of peritoneum, 587
 Sarcinæ in carcinoma of stomach, 239
 Sarcoma of appendix, 434
 of biliary ducts, 850
 fusocellular, 310
 of gall-bladder, 850
 of liver, 801
 of œsophagus, 103
 of pancreas, 677
 of peritoneum, 591
 retroperitoneal, diagnosis of, from perigastric abscess, 543
 of tongue, 67
 Scirrhus carcinoma of stomach, 226
 Scleroderma of mouth, 54
 Scleroma of mouth, 60
 Sclerosis of stomach, 310
 submucous, with chronic gastritis, 310
 Scurvy, intestinal hemorrhage and, 349
 Secretin, 330, 331
 Secretion, digestive, disharmony of, 34
 gastric, factors in, 29
 hormones and, 29
 nervous system and, 25
 salivary, diminution of, 69
 Secretion, salivary, disturbances of, 68
 increase of, 68
 Sensations, gastric, abnormal, 139
 oral, disturbances of, 42
 Sepsis, oral, 41
 Septicæmia in appendicitis, 419
 breath in, 42
 Septicopyæmia, diarrhœa and, 367
 Serositis, multiple, 737
 Shock in acute pancreatitis, 640
 Sialorrhœa, 36, 69
 Sialosis, 69
 Signoidopexy in descent of intestines, 620
 Sitophobia, 164
 definition of, 164
 etiology of, 164
 treatment of, 165
 Skin in ascites, 501, 505
 Sluggishness of bowels, 352
 Smoker's patch, 65
 Soor, 49
 Spasm of œsophagus, 87
 of pylorus, diagnosis of, from carcinoma, 245
 of tongue, 62
 Spine, deformities of, acute dilatation of stomach and, 282
 diseases of, diagnosis of, from gastric ulcer, 207
 Spirochaetes in mouth, 41
 Splanchnoptosis, 36, 599
 abdomen in, 607
 backache in, 607
 belt test of Glénard in, 607
 cachectic, 608
 cardialgia in, 607
 constipation in, 607
 descent of intestines in, 612
 of pancreas in, 613
 of spleen in, 613
 diagnosis of, 613
 displacement of uterus and, 606
 etiology of, 601
 frequency of, 604, 606
 gallstones in, 608
 gastro-intestinal, 608
 hepatic, 608
 indigestion in, 607
 lithæmic, 608
 nausea in, 607
 neurasthenia in, 607
 pain in, 607
 pathogenesis of, 601
 prognosis of, 615
 symptoms of, 606
 treatment of, 615
 mechanical, 618
 medicinal, 617
 general, 616
 operative, 618
 prophylactic, 616
 vomiting in, 607
 Spleen in abscess of liver, 754

- Spleen in abscess of liver, subphrenic
 abscess and, 546
 in acute non-suppurative hepatitis,
 744
 yellow atrophy of liver, 720, 722
 in amyloid liver, 715
 anatomy of, 600
 in biliary cirrhosis, 792, 793
 in chronic perihepatitis, 739, 741
 descent of, 613-
 diagnosis of, 615
 symptoms of, 613
 treatment of, 620
 diseases of, acute peritonitis and, 516
 enlarged, diagnosis of, from carci-
 noma of stomach, 244
 in hæmochromatosis, 783
 perforation of, in gastric ulcer, 199
 in portal cirrhosis, 770
 rupture of, hæmoperitoneum and, 511
 in suppurative cholangitis, 813
 pylephlebitis, 709
 in thrombosis of portal vein, 707
 Splenic veins, thrombosis of, in suppu-
 rative pancreatitis, 649
 Sprue, 440
 diarrhœa in, 441
 etiology of, 441
 fæces in, 441
 mouth in, 441
 pathology of, 441
 prognosis of, 442
 tongue in, 441
 treatment of, 442
 Squamous-celled carcinoma of stomach,
 226
 Staphylococci in appendicitis, 407
 in diarrhœa, 368
 in intestines, 339
 Staphylococcus pyogenes in mouth, 40
 Steapsin, 330
 Steatorrhœa, calculi of pancreas and, 661
 in cysts of pancreas, 672
 in diseases of pancreas, 621
 Stenosing tuberculosis of intestines, 437
 Stenosis, cardiac, gastric ulcer and, 202
 of duodenum, duodenal ulcer and,
 392
 of intestines, congenital, 458
 constipation and, 356
 of œsophagus, diagnosis of, 91
 from spasm, 88, 96
 non-malignant, 94
 of pylorus, 296. *See* Pylorus, sten-
 osis of.
 congenital, hypertrophic, 303
 hypertrophic, 310
 diagnosis of, from carcinoma
 of stomach, 242, 245
 Stercoræmia, 357
 Stercoral ulcers of intestines, 396
 Stomach, achylia of, 159
 adenocarcinoma of, 226
 Stomach, anatomy of, 600
 atony of, 166
 appetite in, 167
 breath in, 42
 cholelithiasis and, 167
 constipation in, 167
 definition of, 166
 diagnosis of, 168
 from continuous hyperse-
 cretion, 127
 from gastrectasia, 168
 from hyperacidity, 117
 from nervous dyspepsia, 173
 dyspeptic asthma in, 168
 etiology of, 166
 gastric contents in, 167
 intestines and, 168
 nervous dyspepsia and, 172
 neurasthenia and, 167
 prognosis of, 169
 pyrosis in, 167
 symptoms of, 167
 treatment of, 169
 diet in, 169, 170
 tuberculosis and, 167
 carcinoma of, 220
 acetic acid in, 237
 acetone in, 232
 age in, 221
 albuminuria in, 232
 alcohol and, 223
 amœbæ in, 239
 anæmia in, 232
 ascites in, 228
 auscultation in, 235
 auto-inoculation and, 223
 Boas-Oppler bacilli in, 238
 bowels in, 231
 butyric acid in, 237
 carcinoma of pancreas and, 678
 circumscribed, 225
 constipation in, treatment of,
 248
 course of, 246
 definition of, 220
 developing upon gastric ulcer,
 201
 diacetic acid in, 232
 diagnosis of, 239
 from achylia gastrica, 161,
 242
 from Addison's disease, 242
 from aneurism of abdomi-
 nal aorta, 244
 from atrophy of mucous
 membrane, 242
 from carcinoma of duode-
 num, 243
 of liver, 244
 from chronic gastritis, 241,
 274
 from cirrhosis, 318
 from enlarged spleen, 244

- Stomach, carcinoma of, diagnosis of, from
- extragastric growths, 243
 - from gastralgia, 137
 - from gastric ulcer, 205, 241
 - from gastroptosis, 615
 - from gumma of liver, 244
 - from hæmatemesis, 324
 - from hypertrophic stenosis of pylorus, 242, 245
 - from movable kidney, 244
 - from neuroses, 241
 - from nervous dyspepsia, 173
 - from pancreas, 244
 - from perigastritis, 243
 - from pernicious anæmia, 242
 - from portal cirrhosis, 781
 - from spasm of pylorus, 245
 - from tuberculosis of peritoneum, 243
 - from tumors of colon, 244
 - of gall-bladder, 244
 - of omentum, 243
 - of peritoneum, 243
 - when tumor exists, 242
 - from ulcer of pylorus, 242, 244
 - of tumor, 240
- diarrhœa in, treatment of, 248
- diet in, 247
- diffuse, 225
- digestive symptoms of, 229
- dissemination of, 226
- duration of, 246
- etiology of, 221
- exploration in, 249
- extension of, 226
- ferments in, 238
- fever in, 233
- flagellata in, 239
- functional signs of, 235
- gastrectomy in, 251
- gastric analysis in, 235, 238
 - disease and, 222
 - ulcer and, 222, 229
- gastro-enterostomy in, 252
- gastrostomy in, 253
- hæmatemesis in, 230
- heredity in, 222
- hyperacidity in, 236
- incidence of, 220
- inspection in, 233
- jejunostomy in, 253
- lactic acid in, 237
- latent, 228
- medullary, 226
- metastases in, 227
- morbid anatomy of, 223
- motor insufficiency in, 235
- multiple, 224
 - thrombi in, 228
- nutrition in, 232
- Stomach, carcinoma of, "occult bleeding"
 - in, 231, 238
- onset of, 228
- pain in, 231
 - treatment of, 248
- pathology of, 223
- peptonuria in, 232
- perforation in, 227
- peritoneal, 228
- prognosis of, 247
- pus in, 231
- rigidity in, 235
- sarcinæ in, 239
- scirrhus, 226
- secondary, 228
- secretory power in, 236
- sex in, 221
- signs of, 233
- simplex, 226
- site of, 223
- social position and, 223
- squamous-celled, 226
- stomach contents in, 235
- symptoms of, 228
- trauma and, 223
- treatment of, 247
 - medical, 247
 - surgical, 249
 - methods of, 251
 - palliative, 252
 - remote results of, 252
 - special considerations in, 250
- trichomonas in, 239
- tumor in, 233
- urine in, 231
- vomiting in, 229
- weakness in, 232
- yeast cells in, 239
- cardia of, carcinoma of, diagnosis of, 245
 - symptoms of, 246
 - treatment of, 247
- cirrhosis of, 271, 310
 - abdomen in, 317
 - anorexia in, 316
 - ascites in, 317
 - cachexia in, 317
 - cardia in, 314
 - circumscribed, 316
 - constipation in, 317
 - course of, 318
 - definition of, 310
 - diagnosis of, 317
 - from carcinoma of stomach, 318
 - from gastric ulcer, 318
 - from syphilis, 318
 - from tuberculous peritonitis, 318
 - duration of, 318
 - etiology of, 313
 - gastric contents in, 317

- Stomach, cirrhosis of, hæmatemesis in, 316
- histology of, 315
 - history of, 311
 - jaundice in, 317
 - lymphatics in, 315
 - morbidity anatomy of, 314
 - mucosa in, 314
 - muscularis in, 315
 - pain in, 316
 - pathogenesis of, 313
 - peristalsis in, 317
 - pylorus in, 314
 - submucosa in, 315
 - symptoms of, 316
 - treatment of, 318
 - tumor in, 317
 - urine in, 317
 - vomiting in, 316
- dilatation of, acute, 24, 280
- abdominal operations and, 281
 - debilitating conditions in, 280
 - deformities of spine and, 282
 - diagnosis of, 283
 - dietetic errors and, 282
 - dyspnoea in, 283
 - etiology of, 280
 - morbidity anatomy of, 283
 - pain in, 283
 - pancreas in, 282
 - pressure of kidney and, 282
 - prognosis of, 284
 - signs of, 283
 - spasm of cardia and, 282
 - of pylorus and, 282
 - symptoms of, 283
 - traction of duodenum and, 281
 - trauma and, 281
 - treatment of, 284
 - vomiting in, 283
- atonic, diagnosis of, from hypertonic, 295
- breath in, 42
- chronic, 284
- anæmia in, 290
 - bowels in, 289
 - course of, 298
 - diagnosis of, 294
 - from chronic gastritis, 295
 - from dilated colon, 295
 - from gastric crises, 295
 - from gastroptosis, 295
 - from hypersecretion, 295
 - from nervous dyspepsia, 295
- duration of, 298
- emaciation in, 289
- gastric contents in, 293
- Stomach, dilatation of, chronic, gastroenterostomy in, 303
- gastropliation in, 303
 - inflation in, 292
 - inspection in, 292
 - pain in, 289
 - palpation in, 292
 - pathological anatomy of, 288
 - percussion in, 292, 293
 - physical signs of, 292
 - prognosis of, 299
 - pyloroplasty in, 303
 - resection of pylorus in, 303
 - skin in, 289
 - symptoms of, 289
 - tetany in, 290
 - treatment of, 292
 - treatment of, 299
 - diet in, 300
 - mechanical, 301
 - medicinal, 302
 - prophylactic, 299
 - surgical, 303
 - urine in, 290
 - varieties of, 284
 - vomiting in, 289
- definition of, 278
- diagnosis of, from dilatation of œsophagus, 295
- idiopathic, 285
- diagnosis of, from obstructive, 295
 - etiology of, 285
 - pathological anatomy of, 288
- nephroptosis and, 611
- diseases of, acute peritonitis and, 516
- constipation and, 354
- functional, 105
- organic, 175
- dyspepsia of, 170
- enlargement of, 278. *See* Gastrectasis.
- eructations from, 143
- fibroid induration of, 310
- hour-glass, gastric ulcer and, 202
- etiology of, 203
 - symptoms of, 203
 - treatment of, 220
- hyperacidity of, 111
- hyperæsthesia of, 138
- hypersecretion in, 122
- hypertrophy of, with induration, 310
- hyperchlorhydria of, 111, 155
- incontinence of pylorus of, 165
- inflammation of, 253. *See* Gastritis.
- lesser curvature of, carcinoma of, diagnosis of, 245
- nervous control of, 105
- neurones of, 105. *See* Gastric neurones.

- Stomach, perforation of, in acute pancreatitis, 643
 in suppurative pancreatitis, 649
 peristaltic unrest of, 154
 in portal cirrhosis, 770
 regurgitation from, 141
 sclerosis of, 310
 spasm of cardia of, 150
 pylorus of, 152
 subacidity of, 155
 syphilis of, diagnosis of, from cirrhosis, 318
 tumors of, diagnosis of, from carcinoma of liver, 799
 within, diagnosis of, 244
 regional, 245
 ulcer of, 175. *See* Gastric ulcer.
 diagnosis of, from cholelithiasis, 842
 from gastralgia, 137
 from gastric hyperæsthesia, 139
 from hyperacidity, 117
 from nervous dyspepsia, 173
 from portal cirrhosis, 781
 hyperacidity and, 114
 varices of, diagnosis of, from hæmatemesis, 323
- Stomatitis, aphthous, 46
 confluent, 47
 breath in, 42
 catarrhal, 45
 gangrenous, 48
 bacteriology of, 48
 infectious diseases and, 48
 symptoms of, 49
 treatment of, 49
 gonococcal, 51
 herpetic, 51
 mercurial, 48
 mycotic, 49
 ulcerative, 47
 bacteriology of, 47
 symptoms of, 47
 treatment of, 48
- Stomatopharyngitis herpetica, 51
- Strangulation of bowel, intestinal hemorrhage and, 350
 of intestines, 443
- Streptococci in appendicitis, 407
 in diarrhœa, 368
 in intestines, 339
- Streptococcus brevis in mouth, 40
 pyogenes in appendicitis, 407
 in mouth, 40
- Stricture of intestines, 443
 obstruction due to, 457
 of œsophagus, non-malignant, 94
- Subacidity, 155. *See* Hypochlorhydria.
- Subphrenic abscess, 545
 definition of, 545
 gaseous, 555. *See* Subphrenic pneumothorax.
- Subphrenic abscess in gastric ulcer, 189
 without gaseous contents, 545
 abscess of Fallo-
 pian tubes
 and, 547
 of kidneys
 and, 547
 of liver and,
 546
 of pancreas
 and, 547
 of spleen and,
 546
 appendicitis and,
 545
 bacteriology of,
 552
 diagnosis of, 553
 from empy-
 ema, 554
 from pleural
 effusion,
 554
 Fürbringer's
 sign in, 555
 Pfehl's sign
 in, 555
 duodenal ulcer
 and, 547
 duration of, 553
 empyema and, 547
 etiology of, 545
 extraperitoneal,
 551
 fever in, 553
 gastric ulcer and,
 546
 intraperitoneal,
 548
 morbid anatomy
 of, 548
 onset of, 552
 pain in, 552
 pericolic abscess
 and, 547
 physical signs of,
 552
 prognosis of, 555
 pulmonary embol-
 ism in, 553
 symptoms of, 552
 traumatism and,
 548
 treatment of, 555
 tuberculosis of
 spine and, 547
- pneumothorax, 555
 abdomen in, 558
 age in, 557
 appendicular abscess and, 557
 complications of, 558
 definition of, 555
 diagnosis of, 558

- Subphrenic pneumothorax, diagnosis of,
 from diaphragmatic hernia, 559
 from pleural effusion, 559
 duodenal ulcer and, 556
 emphysema in, 558
 etiology of, 556
 fever in, 558
 gastric ulcer and, 556
 hydatid cyst of liver and, 556
 morbid anatomy of, 557
 onset of, 557
 pain in, 558
 parotitis in, 558
 physical signs of, 558
 prognosis of, 559
 sex in, 557
 symptoms of, 557
 treatment of, 559
 ulcer of colon and, 557
 vomiting in, 558
- Succus entericus, 331
- Sugar in carcinoma of stomach, 232
- Sugar-iced liver, 737
- Suppuration of mesentery, 542
- Suppurative cholangitis, 811
 cholecystitis, 819
 hepatitis, 745
 pancreatitis, 646
- Suprapapillary carcinoma, 479
- Syncope in spasm of œsophagus, 88
- Syphilis, acute yellow atrophy of liver and, 719
 chronic pancreatitis and, 652
 perihepatitis and, 738
 diagnosis of, from portal cirrhosis, 780
 gastric ulcer and, 180
 of intestines, 439
 of liver, 795
 of mouth, 58
 of œsophagus, 86
 of pancreas, 656
 portal cirrhosis and, 761
 of salivary glands, 72
 of stomach, diagnosis of, from cirrhosis, 318
- T**
- TARTAR, 72
 treatment of, 73
- Taste, disturbances of, 43
- Teichmann's test for "occult bleeding," 193
- Telangiectases of skin in jaundice, 694
- Tenesmus in intussusception, 462
 in volvulus, 464
- Tetany in chronic dilatation of stomach, 290
 treatment of, 292
 gastric ulcer and, 202
- Thrombophlebitis of hepatic veins, 704
- Thrombosis in acute pancreatitis, 643
 gastric ulcer and, 182
 of hepatic artery, 701
 veins, 703
 of mesenteric arteries, 487
 of portal vein, ascites and, 500
 mesenteric thrombosis and, 488
 septic, appendicitis and, 411
- Thrombotic ulcer of intestines, 395
- Thrush, 49
 diagnosis of, 51
 symptoms of, 50
 treatment of, 51
- Tinea circinata of mouth, 54
- Tongue anomalies of, 61
 atrophy of, smooth, 59
 bifid, 61
 black, 62
 bleeding, 62
 carcinoma of, 67
 in constipation, 357
 in diarrhœa in childhood, 385
 doubling of, 61
 fibroma of, 67
 frœnum of, deformities of, 62
 ulcer of, 63
 geographical, 63
 hemorrhage from, 62
 hyperkeratosis of, 62
 inflammation of, 63
 melanotrichia of, 62
 painful, 64
 paralysis of, 62
 sarcoma of, 67
 scrotal, 62
 spasm of, 62
 in sprue, 441
 sucking, 62
 swallowing, 62
 tuberculosis of, 58
 diagnosis of, 58
 treatment of, 58
 tumors of, 67
 amyloid, 67
 varicose veins of, 62
- Tonsillitis, breath in, 42
- Toxic gastritis, 261. *See* Gastritis, toxic
- Transverse colon, anatomy of, 600
- Trauma, acute dilatation of stomach and, 281
 carcinoma of stomach and, 223
- Traumatic cysts of pancreas, 668
- Traumatism, chronic peritonitis and, 561
 subphrenic abscess and, 548
- Trichiniasis, gastric ulcer and, 181
- Trichomonas in carcinoma of stomach, 239
- Trophic ulcers of intestines, 397
- Tropical liver, 726, 743
- Trypsin, 330
- Tubercle bacillus, chronic perihepatitis and, 738
 in intestines, 339.

- Tubercle bacillus in mouth, 40
 Tuberculosis of appendix, 432
 atony of stomach and, 167
 of Fallopian tubes, tuberculosis of
 peritoneum and, 569
 gastric ulcer and, 181
 of intestines, 434. *See* Intestines,
 tuberculosis of.
 chronic hyperplastic, 562
 of gums, 58
 of liver, 795
 of mouth, 58
 of œsophagus, 86
 of pancreas, 656
 of peritoneum, 567. *See* Perito-
 neum, tuberculosis of.
 diagnosis of, from appendicitis,
 427
 from carcinoma of stomach,
 243
 from chronic perihepatitis,
 742
 portal cirrhosis and, 761, 779
 pulmonary, in tuberculosis of perito-
 neum and, 574
 of salivary glands, 71
 of spine, subphrenic abscess and, 547
 of tongue, 58
 Tubular diarrhœa, 491
 Tumor in acute pancreatitis, 642
 in appendicitis, 417
 of appendix, 433
 of biliary ducts, suppurative cholan-
 gitis and, 812
 in carcinoma of intestines, 482
 of pancreas, 679
 in cirrhosis of stomach, 317
 of colon, diagnosis of, from carci-
 noma of stomach, 244
 in congenital stenosis of pylorus, 307
 fœcal, diagnosis of, from nephro-
 ptosis, 615
 of gall-bladder, diagnosis of, from
 carcinoma of stomach, 244
 in gastric ulcer, 195
 of intestines, benign, 485
 malignant, 476
 in intussusception, 461
 of lips, 45
 of liver, benign, 801
 malignant, 795
 of mouth, 52
 of omentum, diagnosis of, from car-
 cinoma of stomach, 243
 pelvic, constipation and, 356
 of peritoneum, 579
 malignant, 591
 primary, 591
 secondary, 594
 non-malignant, 579
 cystic, 583
 solid, 579
 phantom, 341
 Tumor of salivary glands, 74
 in suppurative pancreatitis, 618
 of tongue, 67
 in tuberculosis of peritoneum, 573
 in volvulus, 464
 Tylosis linguæ, 65
 Tympanites, 340. *See* Intestines, gas-
 cous distention of.
 in acute peritonitis, 525
 diagnosis of, from acute peritonitis,
 532
 from ascites, 507
 Typhoid bacillus in acute cholecystitis,
 817, 818
 in chronic peritonitis, 564
 in intestines, 339
 fever, diagnosis of, from appendi-
 citis, 425
 from cholera nostras, 376
 from intestinal obstruction,
 451

U

- ULCERS of colon, 397
 subphrenic pneumothorax and,
 557
 duodenal, 207, 389. *See* Duodenal
 ulcer.
 of frenum of tongue, 63
 gastric, chronic, 175. *See also* Gas-
 tric ulcer.
 diagnosis of, from gastralgia, 137
 from gastric hyperæsthesia,
 139
 from hyperacidity, 117
 from nervous dyspepsia, 173
 etiology of, 26
 function and, 26
 hyperacidity and, 114
 intestinal hemorrhage and, 350
 peptic, 175
 perforating, 175
 round, 175
 of intestines, catarrhal, 395
 decubital, 396
 embolic, 395
 follicular, 396
 leukæmic, 397
 stercoral, 396
 thrombotic, 395
 trophic, 397
 uræmic, 394
 of œsophagus, 84
 decubitus, 84
 peptic, 84
 pressure, 84
 uræmic, 85
 peptic jejunal, 394
 of pylorus, diagnosis of, from carci-
 noma of stomach, 242, 244
 of stomach, 175. *See* Gastric ulcer.

- Ulcus ventriculi, 175
 Ulceration of bowel, 388
 Ulcerative cholecystitis, chronic, 820
 oesophagitis, 80
 stomatitis, 47. *See* Stomatitis, ulcerative.
 Umbilicus in tumors of peritoneum, 596
 Urachus, cysts of, 589
 Uræmia, breath in, 42
 diagnosis of, from acute peritonitis, 534
 from gastric ulcer, 205
 Uræmic ulcer of intestines, 394
 of oesophagus in, 85
 Urea-forming function of liver, 687
 Urination in nephroptosis, 610
 Urine in abscess of liver, 753
 in active congestion of liver, 727
 in acute enteritis, 375
 non-suppurative hepatitis, 744
 peritonitis, 528
 yellow atrophy of liver, 721, 722
 in appendicitis, 418
 in carcinoma of stomach, 231
 in chronic dilatation of stomach, 290
 gastritis, 272
 in cirrhosis of stomach, 317
 in congenital stenosis of pylorus, 307
 in continuous hypersecretion, 126
 in enterocolitis, 385
 in fat necrosis, 633
 in gastric ulcer, 188
 in gastrosuccorrhœa, 129
 in hyperacidity, 117
 in jaundice, 695
 in nephroptosis, 610
 in passive congestion of liver, 734
 in periodic hypersecretion, 123
 in portal cirrhosis, 779
 in suppurative pancreatitis, 649
 Urobilinuria in jaundice, 696
 in portal cirrhosis, 779
 Urticaria in chronic dilatation of stomach, 289
 of mouth, 53
 Uterine colic, diagnosis of, from intestinal colic, 348
 Uterus, displacement of, constipation and, 356
 splanchnoptosis and, 606
 fibromyoma of, diagnosis of, from hydatid cysts of peritoneum, 584
 myoma of, diagnosis of, from nephroptosis, 615
- V**
- VALVE of Gerlach, 403
 Valvular disease of heart, intestinal hemorrhage and, 350
 Veins in ascites, 501, 505
 of appendix, 404
 hepatic, diseases of, 703
 portal, diseases of, 705. *See* Portal vein.
 in tumors of peritoneum, 596
 Venous stasis, diagnosis of, from hæmatemesis, 323
 Vitiligoidea in jaundice, 694
 Volvulus, 462
 abdomen in, 464
 in carcinoma of intestines, 483
 constipation in, 464
 diarrhœa in, 464
 etiology of, 462
 of omentum, 469
 pain in, 464
 pathological anatomy of, 463
 prognosis of, 464
 seats of, 463
 symptoms of, 464
 tenesmus in, 464
 tumor in, 464
 vomiting in, 464
 Vomiting in achylia gastrica, 159
 in acute cholecystitis, 821
 dilatation of stomach, 283
 enteritis, 374
 gastritis, 258
 pancreatitis, 640
 perihepatitis, 736
 peritonitis, 522, 526
 yellow atrophy of liver, 722
 in appendicitis, 415
 in carcinoma of stomach, 229
 central, 145
 cerebrospinal, 145
 in cholelithiasis, 833
 in cholera nostras, 375
 in chronic dilatation of stomach, 289
 interstitial pancreatitis, 655
 in cirrhosis of stomach, 316
 in congenital hypertrophic stenosis of pylorus, 306
 in constipation, 357
 in continuous hypersecretion, 126
 in cysts of pancreas, 672
 diagnosis of, from regurgitation, 142
 in diarrhœa in childhood, 384, 385
 in diverticula of oesophagus, 93
 in duodenal ulcer, 209, 391
 in enterocolitis, 384
 fæcal, causes of, 448
 in fatty liver, 714
 in gastric ulcer, 190
 treatment of, 214
 in hour-glass stomach, 203
 in hyperacidity, 116
 hysterical, diagnosis of, from gastric ulcer, 205
 in intestinal diverticula, 475
 indigestion, 344
 obstruction, 448
 in intussusception, 461
 juvenile, 147

Vomiting in meteorism, 342
 nervous, 144
 definition of, 144
 diagnosis of, 148
 etiology of, 145
 eye-strain and, 147
 gastralgia, 136
 prognosis of, 148
 proper, 146
 treatment of, 148
in periodic hypersecretion, 123
periodical, 146
 in children, 147
 diagnosis of, from hypersecretion, 124
in phlegmonous gastritis, 260
in portal cirrhosis, 773
psychic, 147
in pylorospasm, 153
reflex, 147
 in pregnancy, 147
in splachnoptosis, 607
in subphrenic pneumothorax, 558
in suppurative pancreatitis, 648
in toxic gastritis, 262

Vomiting in volvulus, 464
 of omentum, 469
Vomitus cruentus, 319

W

WANDERING rash of tongue, 63
Weil's disease, 690
Wirsung, duct of, calculi of pancreas and, 659

X

XANTHELASMA in jaundice, 694
Xanthoma in jaundice, 694
Xanthopsia in jaundice, 697
Xerostomia, 69

Y

YEAST cells in carcinoma of stomach, 239
Yellow fever, intestinal hemorrhage and, 349



0032456140

DATE DUE

[illegible]

